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EDITORIAL

BRITAIN'S FESTIVAL OF 1951

He who knows the past can control the future, so let us cast our eyes back over the last century. The great exhibition of 1851 was designed to demonstrate to the world the greatness of our island folk. It owed its origin to the vision and initiative of Albert, the Prince Consort. At that time the so-called industrial age had just begun to swing into full momentum, and the people were to witness great changes in the mode and tempo of life such as would surpass that of all previous record. Mechanical power was to replace man power, sweat and toil to be alleviated, and the harnessing of the wind and water was to be no longer necessary.

The machine age was born and great manufacturing centres arose around the sources of raw material, and a shadow of smoke came over the land. Agricultural life became of secondary importance and the great migration to the towns began, for man has to work that he may eat. A new conception of values was abroad and the strength and power of a country was measured according to its industrial prosperity.

Britain had provided the men whose inventive genius made these things come to pass, and others who followed urged on the factory wheels. Britain was in the vanguard of all this development and so-called progress, competitors were outdistanced, and her industrial products were pre-eminent in the world.

She reigned supreme, and the exhibition of 1851 reflected her renown as a mirror to the world. When we review this past century it is good to note that our people did not entirely concentrate all their attention on purely material

things and in the accumulation of personal wealth. The arts and sciences and the humanities were not neglected, and the standard of life of the ordinary man was enhanced far beyond the dreams of his forefathers. Great men and women walked the stage of life, men who endeavoured and succeeded in their efforts to make life better. Lister, Simpson, Nightingale, Bell and Lodge come easily to mind. and there were many others, and their work was often financed and sponsored by the great industrialists.

It is sad to relate that in one field of endeavour such was not the case. Agriculture and its associated arts and sciences were often left behind.

It may be difficult at the present time to visualise the deplorable depths to which agriculture had sunk in the 1880-90 decade. Poverty prevailed in the countryside, farms were vacated by families which had been rooted in the soil for generations, and the men-folk had fain to seek occupation in the towns. It is right, at this point, that we should pay credit to those who deserve it. It was the public spirit and often the self-denial of the great land-owners which kept the agricultural torch alight. So agriculture and animal husbandry continued to exist, with occasional intermissions of prosperity, such as during two world wars, until quite recent times. Now, it is strange that the thing which was responsible for all this decline in our indigenous agrarian economy is, in turn, responsible for the present recrudescence.

The reason is not far to seek. Whilst Britain was the supreme industrial centre of the world, local agriculture was of little importance, for the products of the earth rolled constantly in, in exchange for manufactured products.

Food was plentiful and cheap.

The time has come when our erstwhile customers can supply many of their own wants, and they are loath to supply our needs of food. Indeed, in many cases surplus for export does not exist. We are therefore compelled to rely more and more on our own food resources. We are in a dilemma. It is essential that there be no let-up in industry, for survival as a first-class nation depends upon that factor. We must keep our industries going at top level.

Our farms can be no longer relegated to oblivion—they must produce more and more food. We must make two blades of grass grow where one grew before.

The present Festival of Britain, like that of a century ago, can be a mirror to the world. It can, and will, bring prosperity in its train, but only if we take full advantage of the opportunity afforded.

The veterinary profession has a great part to play; it is an essential factor in a prosperous agrarian economy, and the farmers look to its members for advice and help in their great task.

Animal disease can, and must, be controlled; in many cases it can be rooted out. The last few decades have witnessed great advance in our knowledge of disease and its control and cure. Let everyone play his full part. If they do so then this festival of 1951 will mark an epoch in our island story.

XUM

SPECIAL ARTICLE

HISTORY OF VETERINARY PROFESSION 1851-1951

A.—The state of the Veterinary Art in Britain at the date of the Great Exhibition of 1851

By J. T. EDWARDS, D.Sc., M.R.C.V.S.

"To be ignorant of what has occurred before our time, is ever to remain in a state of childhood."—CICERO.
"History has been spoken of as a compound of poetry and philosophy, and it is easy to prove that there is much of both in the history of veterinary medicine."—JOHN GAMGEE (1867) [*Veterinarian, 40,* 754].

I

"GRAND as the Great Exhibition is, admirable as it is, and even useful as it may turn out to become, medicine can boast but little of the part it has played in the Crystal Palace, and veterinary medicine still less. . . ."

So wrote William Percivall (1792-1854), then "the ablest man in the profession," editorially in the *Veterinarian* in its issue of October 1, 1851, a date when the Great Exhibition, which had opened on May 1, was drawing to its close.

And there was much else in that editorial article—from which we need again to quote—to make one wonder whether, after all, anything worth the attention of the present generation of veterinarians is to be derived from a special study of the significance of the "Great Exhibition" as an episode and of the year 1851 as a landmark. The great landmarks in the history of the veterinary profession in Britain are definitely four in number: first, the founding of veterinary education in 1791; secondly, the Charter of 1844; thirdly, the Veterinary Surgeons Act of 1881; and, fourthly, the Veterinary Surgeons Act of 1948. There are several other notable eminences which, for the moment, we need not pause to contemplate. But, having regard to what Percivall wrote towards the close of the Great Exhibition of 1851—we may be sure, in all sincerity, but with the limitations in perception such as we have come to know them to be—one would be sorely tempted to dismiss lightly the subject we have been invited to treat upon. Indeed, there is so very little on record concerning the participation of the veterinary profession in the Great Exhibition that it could all be readily reproduced within the compass of a short article, leaving the reader mildly interested, but not enlightened.

The truth is that the historic event of the Great Exhibition marked a dividing line in the evolution of the whole body of mankind which could not but have a profound effect upon every possible human activity, including—and by no means the least profound—upon that body corporate whose members only a few short years before, in 1844, had become recognised by Royal Charter as belonging to a profession. That dividing line marked the final triumph of the Industrial Revolution.

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To say what we are about to say amounts perhaps to an overstatement. Since the human race had appeared as a species apart, half a million or more years ago, it gained gradually, and often precariously, an ascendancy over other species (confronted as these were meanwhile with the necessity of adaptation to periodic climatic and terrestial changes) by its ingenuity in the use of simple tools. That same human race now, in 1851, during the course of the preceding few thousand years, and especially within the course of the preceding two hundred years, had progressed so far in its dexterity in devising tools that henceforward these selfsame tools were definitely to establish a mastery over man himself.

Anyhow, it is, at this date, a melancholy reflection that at the Great Exhibition of 1851—with all that was crying aloud for solution at that time of violent transition, in the shape of a transformed agriculture, immense public health problems, and the opening up of a vast empire overseas—all the veterinary profession saw fit to exhibit as an earnest of its own competence was some tools in which at the time it took a vain delight—namely, mainly horseshoes, turned and shaped and polished up in various ways according to the whims and fancies of their proud designers. There were, too, on view a few cases of instruments, of which a very fine sample is to be found in the case of equine dental instruments exhibited by T. W. Gowing (?-1886), "a quiet observant man with a great aptitude for mechanics" and "one of the best practitioners in London," who had qualified from the London College in 1847. That case, which won a prize at the Great Exhibition, is still on view in Professor McCunn's room at that College.

There were shown, we learn, a remarkable array of horse carriages, ranging from those used in trade and commerce to those in which notably the rising middle classes could display their opulence in their rivalry for ascent to social recognition at the level of the upper, land-owning, classes, whose power as a ruling caste in the country had been doomed to eventual displacement with the passing of the great Reform Act of 1832, the standards of domestic and civic conduct in that middle class being austerely set by the young Queen herself and her worthy Consort, whose own conception it had been to display his adopted country to its citizens and to the world within the architectural design of Paxton's huge glasshouse in Hyde Park, fittingly christened at the time the "Crystal Palace," and the nosological and cultural progenitor of those tabernacles of the present age, the "Picture Palace" and "Palais de Danse."

An exhibit in the agricultural section in front of which rural visitors stood agape was one which had been imported from Canada in the shape of a reaping machine, then reputed to be the original invention of a citizen of that country, McCormick, but which had been anticipated by a machine embodying all the essentials of the modern reaper by John Common of Denwick, Northumberland, as early as 1812—popular hostility to it, however, proving so great that he had to drop working on it. Later, in 1822, Common's machine was improved upon by the Browns of Alnwick, who emigrated to Canada, taking models of

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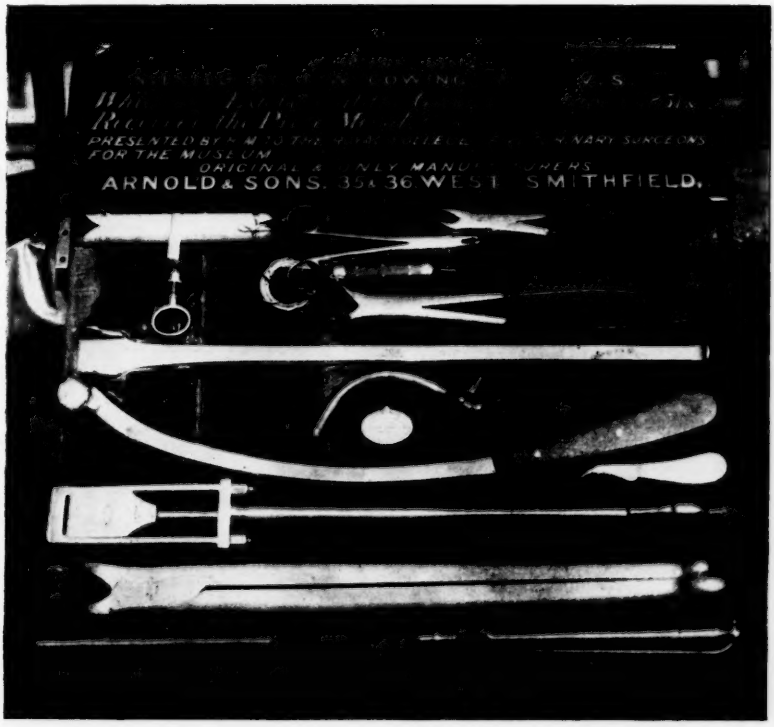

A CASE OF COWING'S INSTRUMENTS
AS EXHIBITED AT THE 1851 EXHIBITION

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Common's reapers with them, so that Common's machine was "almost certainly the father of" McCormick's famous machine. The present writer well recalls the time, just over fifty years ago, when reapers were first introduced on farms in the somewhat remote countryside of his childhood; how, until then, large numbers of skilled scythesmen mowed the hay and corn through neighbours coming to each others' aid at harvest time, all the work of raking and binding being done likewise by the women. With the displacement of these bands, the spirit of neighbourliness commensurately vanished, and the harvests never again knew the same merriment and happiness.

II

The two decades preceding the Great Exhibition saw a revolution in the isolation in which rural pursuits had hitherto been conducted, with the opening up of the railways, the immediately preceding decade being characterised by a "railway mania," during which nearly all the present main-line systems were laid down and which, providentially and unexpectedly, in very large measure solved a widespread and grave problem of unemployment and distress among the labouring classes—a problem which had been harassing legislators since the close of the Napoleonic wars in 1815. That solution brought in its train, however, a new set of problems, concerned with the feeding of the rapidly growing populations in the new towns, the successive Corn Laws designed to protect farmers (while, incidentally, they did not protect the farm labourers) against falling prices through applying restrictions upon importation of foreign grain proving to be a serious handicap against the supply of cheap and sufficient food for the town dwellers. Against formidable opposition, the total repeal of the Corn Laws, as the reader knows, was enacted in 1846 during Sir Robert Peel's great Ministry of 1841-46, "a turning point in British history" and, as we shall have occasion to stress later, "the greatest turning point in the history of the British veterinary profession." That episode demands imperatively a special article to itself at this present stage of our history, as also does the Free Trade in importation of disease that was the consequence of the salutary Free Trade in foods. For, to assess properly the situation in which we find ourselves to-day as a profession, during this present "Festival of Britain" year, in 1951, no sound conclusions whatsoever can be drawn unless and until we have examined carefully the sharp turn of events between the grant of our Charter in 1844 and the date of the Great Exhibition of 1851. We can only now, it can be said, in 1951, and not before now, appreciate that turn of events "in the cool light of history." And, as Sir Frederick Smith said about the Charter episode itself—namely, that a whole volume could be written about it—so another volume could be written about the "turn" between 1844 and 1851. For, irrespective of any episodal celebrations, a distinguished modern historian (Ramsay Muir, "A Short History of the British Commonwealth," 6th Edition) —in general conformity, indeed, with other historians—in a standard treatise separated at the point in question our social history into two books, designated respectively "Book IX, National and Imperial Reconstruction and the Triumph

of Industrialism (A.D. 1815-1852)" and "Book X. The Era of British Complacency; and the Adolescence of Daughter Nations (A.D. 1852-1880)." What could be achieved and was, *in part only*, achieved for our profession in the closing years of the former era was impossible of achievement during the succeeding era. The social "climate" had wholly changed, disastrously for us, but not irremediably. To use a metaphor that is not much more inapt than most metaphors are, the Royal Assent to our Charter of 1844 was a "try," scored as the result of a brilliant concerted movement within the profession, in which the profession stood to gain at once "championship points" over all other professions, and certainly over the medical profession, whose internal affairs were then chaotic in the extreme. And that "try" was scored right under the posts, but the easy kick at goal failed, and only narrowly failed, all through the fault—as we can now see in 1951—of the profession itself. As it was, Bills presented to Parliament in 1853 and 1866 to prevent unqualified practice failed to gain a reading, while the "half measure" that was the Act of 1881, secured at the cost of admitting 188 holders of the Highland and Agricultural Society's certificate to membership and 914 unqualified men to the supplementary register as "existing practitioners" was the most that could be obtained when the "Era of British Complacency" had just drawn to a close. To secure, even later, the Act of 1920, prescribing merely an annual fee on its members to prevent self-extinction, the R.C.V.S. had had to battle patiently for fourteen years, the social climate still being hostile to restriction of the practice of the veterinary art to none other than qualified members of the profession.

This much has been said here in this introductory article, which has for its object merely a portrayal of the state of the veterinary art in Britain in 1851, because so often it has been declaimed, by figures who have been in the limelight of no less eminence in their own day than Fleming and McFadyean, that progress in the science and art of veterinary medicine in Britain has contrasted sharply with that on the continent of Europe because, in the case of the latter, rapid progress was made possible by the active support of the State whereas in the former it had been consistently impeded by the indifference, even the hostility, of the State. Deeper reading of history must now discount that, in part at any rate, as fiction. Sir Robert Peel's great reforming, and, oddly, labelled "Conservative," Ministry of 1841-46 was wholly favourable to the enactment of solid reform in the veterinary profession, and even went out of its way to indicate precisely the manner in which, as the successive events immediately preceding this Festival of Britain year at last clearly show, it ought to set about at once to consolidate its own foundations, to weather all storms later threatening its superstructure. No less favourable was Lord John Russell's Liberal-Whig Administration that immediately followed Peel's great Ministry, that statesman being destined soon to play the leading part in the Royal Agricultural Society of England, founded in 1838 through the initiative of the journalist, William Shaw, a close friend of our William Youatt. If the opportunity had then been seized, the "marriage"

in high estate of veterinary medicine to agriculture would have been for all time assured.

As it soon transpired, however, the signpost or symbol that was the Crystal Palace marked the sudden onset of a new era during which our own healthy progress as a profession in Britain was destined to be becalmed. Only when we emerged from the doldrums of that era, misnamed as it was by writers of the succeeding era as the "Golden Age" (especially of agriculture), could partial progress be made. And then, as it were, it could be made only by stubborn tracking, undertaken by a few helmsmen, like Fitzwygram, Fleming, and McFadyean. The accidental death of Peel in 1850, "the ablest and strongest man" in the political parties, "a sacrificial victim to the Great Exhibition" was no less a tragedy for the veterinary profession (as we can only now discern) than it was for the new political party, the Conservative, he had brought into being out of the fading remains the great historic party, the Tory, which had outlived its usefulness with the passing of the 1832 Reform Act. It was not until the prevailing social and political climate in Britain had been freshened up enough by the events of the 1939-1945 war that rapid forward progress was again made possible—as it could have been made, were it but generally realised at the time, a full century earlier. It is left for the historian of the future to determine whether or not it has been wholly salutary for the barque to be carried along so suddenly by, what seems to many, hurricane force.

That change was symbolised by the Fourteenth International Veterinary Congress held in London in 1949: listeners to the alert and youthful Government spokesman (the Rt. Hon. Hector McNeill) who opened the Congress, with his intimate experience in advocating the British cause in the counsels of the nations, were left in no doubt as to how to reshape their outlook; and subsequent speakers at the plenary sessions, as well as those called upon to register progress at the sectional meetings, provided a symphony whose theme was wholly in harmony. It was that it is to the food and health, bodily and spiritual, of peoples which, now as at the beginning, need to harmonise with the healthy upkeep of other species—and, too, in ever-increasing degree of complexity of relationship as "machines" themselves have evolved in complexity—that the energies of the profession must be bent if it is to subserve its proper and wholly beneficent end. Which end it can subserve, we were again told, better than any other profession, and it can be expressed as that of arresting the tyranny exercised by those "machines" in tempting peoples inexorably towards self-annihilation.

III

To quote again from Ramsay Muir (page 461), the early Victorian Age ushered in a period in which "the supreme interests of the British people were four—business, religion, politics, and sport; imaginative, æsthetic or purely intellectual interests lagged far behind with all but very few. Business came first. . . . But religion came next, among a surprisingly high proportion of the population. . . ." And quoting further from another modern historian

(Gilbert Slater, "The Growth of Modern England," 1st Edition, 1932, page 387), a great writer on the contemporary scene, George Eliot, "is recorded as having discoursed on the three great ideas which had been the guiding stars of humanity, God, Immortality, and Duty, remarking that as belief in God and Immortality faded, belief in and homage to Duty grew stronger."

There was at least one great Englishman of this period who did not fail in his conception of Duty (Slater, *loc. cit.*, pages 378-385), namely, Edwin Chadwick, a disciple of Jeremy Bentham, and a man of untiring zeal and energy in the cause of public hygiene, who, after his exploratory activities as Poor Law Commissioner and his exposure of the horror of living conditions in the towns, secured the passing of the temporary measure which was the first Public Health Act, of 1848. That measure resulted in the appointment of medical officers of health for the towns. "The first report on the health of London, drafted by Dr. [afterwards Sir] John Simon, is one of the most scathing arraignments of its own condition which any city has ever placed before the public." And Lord Shaftesbury, Chadwick's colleague, noted in his diary (May 14, 1852): "*The Times* has taken up the note of the undertakers, the parliamentary agents, the water companies, and the whole tribe of jobbers who live on the miseries of mankind and are hunting the Board of Health through brake and briar and hope to be 'in at the death.'" Among the interests which the Board roused to opposition, Lord Shaftesbury further notes, was "the College of Physicians, and all its dependencies, because of our independent action and singular success in dealing with the cholera, when we maintained and proved that many a Poor Law medical officer knew more than all the flash and fashionable doctors in London." "In 1854 . . . the Commissioners held their farewell dinner. Chadwick was then dismissed from the public service with a pension; paid by the nation henceforward to leave dirt alone. . . ."

It is to the discredit of the British veterinary profession of the time that none of its members (as did the illustrious French veterinarian, Henri Bouley, later uphold Pasteur in his somewhat similar trials) come to the rescue of Edwin Chadwick. If it had done so, it need never have been passed over ignominiously when a substantial and permanent measure of public health control was entered in the Statute Book in the shape of the Public Health Act of 1875. The one man in the profession who had consistently advocated co-operation with both agriculture and other social interests—including, it may be said, the R.S.P.C.A. at its origin—was William Youatt, who was also a temperamental exception to the prevailing early Victorian mood, having, as Leclainche has said of him, "no commercial sense." It was the men endowed with strong commercial sense, combined doubtless with the highest degree of professional sense compatible with it, while meanwhile assuring self-preservation beyond all possible risk,* who had won the day when the Peel and Russell Ministries had exhorted the profession in 1845 and 1846, as a next step

*Leclainche (*loc.-cit.*, pp. 127-129) has given us a vivid and well-documented narrative upon how in the Islamic world from the eighth century A.D. onwards the veterinary art was practised with the most scrupulous "pure" professionalism, shorn of all commercialism, it has ever known; and how, in the outcome, it brought in its train upon its practitioners only too often self-immolation, and even self-ruin.

after gaining the first step, to consolidate their position by establishing "public relations" at the highest level, through practitioners accepting as associates on their Council nominees of the great friendly national interests. One cannot but view now, looking at events from the vantage point of 1951, that the peremptory request of the Minister of Agriculture (the Rt. Hon. R. S. Hudson) to the deputation from the Council in 1945 to turn about on its heels when presenting its studied proposals was but a visitation upon them of the "sins of their forefathers" when, in 1845, a similar deputation appointed to wait upon Peel's Home Secretary (Sir James Graham) gave that able and conciliatory statesman what F. C. Cherry said in his address at the following annual general meeting was "a slap in the face."

IV

Actually, in 1845, the state of veterinary practice in the large towns (see the *Veterinarian, 13,* 288) was rather bad, largely through the imposition of high excise duties on horses, vehicles, and attendants, and the high prices of fodder; but by 1851 the situation had very much improved, mainly through the abolition of the duties, the rapidly increasing wealth of the towns, the great expansion of local horse traffic through the railways, and the decrease in the prices of fodder.

Before 1845, we may well accept the summary given by Leclainche ("Histoire de la Médecine Veterinaire, 1936, page 414) of the state of London practice:—

"Towards the end of the eighteenth century there were to be found a number of fairly well-enlightened men practising in the larger towns, among whom could be counted especially several human surgeons who were discovering that the practice of veterinary surgery was more remunerative than the exercise of their own craft. Several empirics possessing much skill were also to be found. Both of these classes often attained brilliant reputations. Towards 1825, according to W. Rieck, the German veterinary historian ('Veterinärhistorisches Jahrbuch,' 1926, pages 99-120), London was a paradise for veterinarians. About a hundred and twenty could be counted, all of whom had large practices. Most of them also possessed a forge, infirmaries, and a druggist's shop open to all buyers. The most highly reputed were the Fields, whose firm had been founded by Moorcroft (the first British veterinarian to qualify—namely, at Lyons—before the opening of the London School). After them in reputation came the Cherrys, Bracy Clark and his nephew, Charles Clark, Sewell, Henderson, Turner, Hart, Rogers, Goodwin and others. Youatt was associated in practice for nearly thirty years with Delabere Blaine, specialising upon the smaller animals, and their reputation extended far outside the bounds of the metropolis. Youatt ran a subscribing clinic, free treatment of all cases being guaranteed to subscribers.

"Competition between the London practitioners and the London School became embittered (the former protesting that the School had become a private institution, run for profit, and privileged by obtaining fees from subscribers, no

qualified veterinarian being entitled to become a subscriber, and hence the profession had no voice in the management of the College; the teaching duties of the staff, they alleged, occupied but a minor and subsidiary part of their work). Hence, the London School for a long time did not produce a race of men who were substantially different from the ordinary farriers.

"In spite of everything, an elect floated up among the practitioners which organised itself to form the movement which secured the Royal Charter of 1844 constituting those who qualified to practise the science and art of veterinary medicine and surgery into a legally recognised profession."

V

The year 1851 is also a notable event in the history of the profession, because at the beginning of the next year was published the first full Register of Veterinary Surgeons, in which was listed all names of men who qualified from the London and Edinburgh Schools from their foundation until the end of 1851. The total entries were 1,733, of whom there were 1,335 surviving, the oldest member being Edward Bond, the first to pass out of the London School, on April 22, 1794. Within the memory of the writer, the Bonds and Batts of Oxford Street were names held in great deference and respect as an old firm of practitioners, several of the best shoeing smiths in the country (including one with whom the writer was in close contact during his pupilage) having been trained in their forges. It is interesting to note that the copy in the R.C.V.S. library is inscribed with the name of "William Robinson, Orchard House, Tamworth, Staffs," who was President of the R.C.V.S. from January, 1851, to May, 1852, and whose intimate friendship with the great Sir Robert Peel—who sat in Parliament for Tamworth—beyond doubt was in large measure contributory to our "surprising success" (as William Dick testified on his return after the event in a letter to T. W. Mayer) in obtaining our Charter. Readers are enjoined to read in the *Veterinarian* of 1852 an account of the dinner given in honour of this most popular man at the close of his presidency to appreciate in what high esteem the best practitioners of the time were held in exalted places in this country.

The immense labour of preparing that first Register was the outcome of honorary work done by a small committee, the active part being played by an indefatigable and most able young man, the Registrar, W. A. Cherry, the son of F. C. Cherry, who followed Coleman as head of the Army Veterinary Department.

In the Progress Report of the Registration Committee published in 1847, some most revealing information was already forthcoming in response to circulars regarding the manner in which the veterinary art was being practised in this country (*Veterinarian*, 1847, *20*, 270). At that stage of preparation only 425 names of duly certified men had been entered as valid, those members being "but thinly spread over the kingdom." It was added that "those who *assume* the title of Veterinary Surgeon exceeds the number of certified members." "Under the various denominations of *horse-doctors, horse-surgeons, farriers,*

cow-leeches, cattle-doctors, castrators, spayers and gelders, charmers, spell-workers, butty-colliers, water-doctors, and various other local appellations, those who gain a livelihood by the practice of the art far exceed the two other classes combined; and this, too, *without including* chemists and druggists who advertise to prescribe and make up nostrums, or proprietors and vendors of quack medicines; or butchers, coachmen, grooms, ostlers, carters, colt-breakers, bailiffs, herdsmen, shepherds, and others, who pretend to 'doctor' the stock under their immediate care." "Those who *style themselves* Veterinary Surgeons, and those under the various denominations before-mentioned, taken together, in numbers may be safely estimated at somewhere about *six thousand*, while the certificated members do not, as far as the Committee can judge, exceed *fifteen hundred* [an estimate which, in the outcome, did not prove far wrong]. Thus there are, at the lowest estimate, *seven thousand five hundred* who practise the veterinary art as a livelihood." "In those districts where cattle are numerous, the number of the lowest class of the unqualified particularly abound." "It also further appears that the cattle practice is the stronghold and chief maintenance of the lowest and most ignorant class of the untaught." "Those who *assume the style* of Veterinary Surgeon appear for the most part to more particularly attend to horses, and are to be found in the greater number in thickly populated districts, or where horses are numerous."

The facts, therefore, it was claimed, showed some very important points—namely: (1) "the want of veterinary practitioners is extensive"; (2) "unless those who are hereafter destined to become practitioners are properly qualified *to treat* the various maladies to which not alone *horses,* but *cattle, sheep,* and *swine,* are liable, the unqualified and untaught will still retain their places"; and (3) "the manner in which the education of Veterinary Surgeons has been hitherto conducted is very faulty, and calls loudly for revision."

The second Register, published in 1854, contains the names of 1,387 active members; the third, published in 1858, 1,422; the fourth, published in 1863, 1,509; and the fifth, published in 1869, 1,826 (the deceased prior to December 31, 1868, totalling then 962), and for the first time a "County List of Members" is given, in response to a request that the territorial distribution of duly qualified practitioners should become known to the public. Then, the sixth, published in 1871, contains the names of 1,870 active members (the deceased then amounting to 1,002); it also contains separate lists of the veterinary surgeons belonging to the Army Veterinary Department and of those practising in America, Australia, and New Zealand. Though it was decided by the Council in that year, 1871, to publish the Register annually, the next available Register, the seventh, only appeared in 1878, and contains the names of 2,241 active members. It then began to assume its present-day shape, and contained, besides the Charters and bye-laws of the College, a copy of the curriculum of studies, which, to meet the situation reported upon by the Registration Committee in 1847, at last can only be deemed to have been excellent. At this date, it seems it is a pity that in 1894, when the course was extended from three to four years, it was departed from in principle and made, most erroneously, as could be readily proved if continental

experience in that respect had been duly consulted, to conform in its general layout to the contemporaneous medical curriculum. By the supplementary Charter of 1876, it had been directed that "the Register of Members of the R.C.V.S. to be made and maintained shall be styled 'the Register of Veterinary Surgeons' and kept as accurately as possible by the Registrar."

The ninth available copy is dated 1883, and contains a list of 2,553 properly qualified practising members, besides the huge list, as it struck the eye of the writer, of "existing practitioners" who had been brought on the Register under the Act of 1881.

No detail of the subsequent annual Registers need be given, for it is sufficient for our purpose to compare those early lists with the numbers entered in the last published—namely, 1950—Register, which contains the names of 4,929 active members together with those of 222 veterinary practitioners listed in the Supplementary Veterinary Register in compliance with the Act of 1948. If we compare that number with the 1,335 active members listed in the 1851 Register, we might well conclude that the increase in numbers is not more than barely adequate to meet the demands of the country as reported upon by the Registration Committee as far back as 1847. However, there are several other factors that need to be taken into account, such as the much greater mobility and range of practice of individual veterinarians since the advent of motor transport and a greatly improved rural road system.

VI

What the daily life of a successful veterinary surgeon in country practice was like during that early period may be deduced from the vivid narrative left us by Professor Walley (1842-1894), of a member of the famous Stanley family, one of whom was within living memory the possessor of a great horse practice in "the Borough." The Edward Stanley mentioned in the narrative was unanimously elected Chairman of the newly-formed Board of Examiners of the R.C.V.S. immediately after the grant of the 1844 Charter, presumably on account of his reputed knowledge and integrity, but resigned within a year, again presumably because of the unfeigned distaste of that family for anything savouring of "politics," which already, as Smith has stated in his biographical sketch of Professor Simonds (*Hist., 4,* 87), were to lead to "thirty-two years of civil war" which "well-nigh completed the destructive work of Coleman," and which, as we have now stressed, could have been completely averted in 1845 if the profession itself had acted more wisely. The Stanley memoir appeared in this journal.

We have to record, states the Editor of this journal (*Vet. J.*, 1886, *23*, 305), the death of W. T. Stanley, M.R.C.V.S., of Leamington, on August 23, aged 75 years. Mr. Stanley was one of the best-known and most highly respected practitioners in the Midland counties, where he enjoyed, for nearly half a century, the esteem and confidence of all with whom he came into professional contact. He graduated in 1831.

To the remarks we recently made, the Editor adds the following year

(*Vet. J.*, 1887, *24*, 140), with regard to the late Mr. Stanley of Leamington, Professor Walley furnishes the following interesting additions:—

The subject of my sketch, the late Wm. Stanley of Leamington, was, at the time of his death, one of the oldest members of the profession, having, indeed, spent in its active practice as many years as it is the lot of probably the vast majority to live. He graduated in the year 1831, and most of what I have here to record refers to a period now a quarter of a century distant, when I had the privilege of an intimate acquaintance with him, and not infrequently acted as his locum tenens during his prolonged absences from home in the discharge of his professional duties. Being, in 1861, assistant to his brother, the late Mr. E. Stanley, at Birmingham, it was to me both a great pleasure and a great privilege to run over to Leamington for two or three days and give Mr. Stanley all the assistance I could—especially in the way of dispensing; and the preparation of seventy-two dozen balls, with other miscellaneous work, in one day, is not a bad example of the work accomplished.

It would be impossible to refer to the one without, at the same time, thinking of the other, who pre-deceased him some years. Both of them were enthusiasts in their profession, both fond of horses and all connected with them; of extremely active habits, punctual to a degree in the fulfilling of their numerous appointments, and, though hasty in temper, just and honourable in their conduct to all men, and ever ready to make amends for any chance word or act that might have been construed in an offensive light; in each case no pains were spared to gather all the information that was to be had, and in applying it for the benefit of the suffering animal world, and it is recorded of Edward Stanley that on one occasion, in his early life, he disguised himself and made friends with a noted quack in his neighbourhood, with the object of discovering the secret of his reputed success in the cure of Poll-evil; he was successful in his efforts, but was disgusted to find that the empiric's panacea was only the root of the black hellebore plant. Another matter in which the brothers were alike was in their love of hunting, and in their aptitude to utilise its lessons in the practice of their profession, in the way of thoroughly testing all animals submitted to them for examination as to soundness, and in cutting short a journey by following the flight of the crow, instead of diligently plodding over the highways and by-ways of the country in which they practised. This peculiarity is shared by one at least of the descendants of Edward, with whom I have had more than one swinging scamper over hedge, ditch and hurdle, with the object of saving distance.

Considering more closely the life of William Stanley, it may interest the younger members of the profession to know something of the conditions under which a large country practice was conducted in the active part of his career. I have said that he, in common with his brother, was passionately fond of the pig-skin (and saddles were made of the skin of the grunter in those days), and by no chance, if it were possible to do so, would he put his foot inside a carriage or other vehicle. "In the saddle," he used to say, "I feel safe; in a conveyance

I never know what may happen"*; and it may astonish those who have only experience of the present system of practising to learn that he considered a good stretch of work was the covering of sixty miles on one and the same nag; and on returning from each ride he needed nothing more than a wash, a beefsteak or a mutton chop, and a glass of wine, to be ready again for the road if circumstances required it. He always rode animals as nearly as possible thoroughbred, about 15-1 or 2 in height, and, in preference, mares; and, as it behoves all good horsemen to be, was careful of their welfare both at home and abroad.

William Stanley was almost as deaf as a post; nevertheless he could detect, with unerring accuracy, a roarer or a whistler, and he never satisfied himself in the examination of a hunter until he had given him a "burster" over hedge, ditch, and field.

In diagnosis, he possessed wonderful intuitive powers, and in the differentiation of lung affections, deprived as he was of the accessory of the sense of hearing, he seldom went past the head of the patient.

In prognosis, he was as accurate as in diagnosis, and a glance was sufficient to show him that his patient was on the high road to recovery, or the reverse. He was a fearless and brilliant operator, and there are few men living at the present day who would undertake single-handed, as I have known him do, some of the most desperate operations we, as veterinary surgeons, are called upon to perform. Neurotomy,† perhaps, was his favourite operation; always preferring the low operation, he attained to a greater measure of success than is commonly met with. He was one of the few who practised section of the tibial nerve for hock lameness, or of the plantar nerve for incurable lameness in the hind foot; and in a letter of some twenty-four pages, written to me in December, 1884, he relates with great gusto the case of a mare—the property of the late Mr. Robinson, which afterwards came into the hands of Sir Charles Slingsby, and ultimately became the property of Lord Henry Bentinck. This mare met with a severe accident to one of her hind feet, and after the subsidence of the acute symptoms, remained lame and useless. He divided the plantar nerve, with the result that she carried each of the gentlemen mentioned for several seasons in the most brilliant fashion. She cost Lord Henry Bentinck 175 guineas, and after his death was sold at Tattersall's for the sum of 140 guineas; and it was with a chuckle that Mr. Stanley directed attention to the fact that

* This had reference to the dreadful state of the roads during that period, which, though by then they had much improved, made travelling in a wheeled vehicle in rural districts—even within the memory of the oldest still surviving veterinary surgeons—a somewhat hazardous undertaking.

†Towards the end of the eighteenth century, Wm. Moorcroft, the first qualified veterinary surgeon to practise in this country, introduced into his Oxford Street practice an operation for the relief of foot lameness, which for many years had been performed by human surgeons in the treatment of painful neuralgic affections. He did not, however, make any public announcement of this operation, and it was not until Professor Sewell, in a paper read before the Royal Society, claimed originality for it, that Moorcroft—then practising in Calcutta—came forward and undoubtedly proved that he had a prior claim to the discovery. The operation became universal after about the second decade of the nineteenth century (see Smith, F. [1886] : *Vet. J., 24,* 189).

the professional men, who at different times examined her, never detected that the operation of neurotomy had been performed.

In castration, when I knew him, Mr. Stanley preferred the écraseur; but I must confess that, of this operation at least, I was not particularly enamoured, although he was on the whole successful. His anxiety for the recovery of the patients under his care was shown by the eagerness with which he watched for signs of returning health, and the pleasure with which he hailed such signs. I have seen him spend many precious moments in tempting the appetite of a patient with a handful of grass or hay or small quantities of corn, and his delight was unbounded when he succeeded in persuading it, as it were, to take even a single blade of grass from his hand.

The enthusiasm of Mr. Stanley in the practice of his profession remained with him to the end, and a short conversation, which it was my privilege to hold with him a year prior to his death, was to me like travelling over the vista of a distant past, and linking together the times that were and those which are. The pleasure thus afforded had only one drawback—his extreme deafness, and it was painful to be obliged to have recourse to a slate and pencil for the purpose of carrying on our conversation.

A contemporary of Carless, Robinson, Lepper, and Litt, he was in every way as worthy; and although he never came to the front in the political sense, he was always anxious to know what was going on in the political world. In one respect he was ahead of his time, and very many years ago he said good-bye to politics, because those who were then in power refused to join him in an attempt to make the practice of the profession by any other than qualified men penal; and almost the last words he addressed to me were in condemnation of the half-measure (with which we were fain to be content) embodied in the Veterinary Surgeons Act.*

To those whose memory can carry them back to William Stanley's time, I trust this imperfect sketch of his life will be acceptable. To those whose years as practitioners are few, I hope it may be an incentive to emulate his enthusiasm, and to love the profession they have chosen for its own sake.

VII

The Stanley memoir gives us what we may take to be a faithful account of the improvement wrought by the provision of properly-instructed veterinary surgeons for the countryside, a salutary state far different from that obtainable from the " charmers " and " spell-workers " and the rest of their kind mentioned in the 1847 Report of the R.C.V.S. Registration Committee. Of the practices indulged in by that tribe we have again on record a vivid narrative from the pen of Henri Bouley (*Rec. Méd. Vét.*, 1853, *10*, 384), supplementing the publication of two letters written by Voltaire in 1771 and 1775, from Ferney, to Claude Bourgelat, the illustrious founder of veterinary schools in Europe, having opened one school at Lyons in 1761, and another at Alfort, near Paris, two years later. Voltaire, in his second letter, after referring to his own life as being essentially

* The Act of 1881, the defects of which in this respect were only remedied with the passing of the Act of 1948.

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that of a farmer, concludes his second letter by saying, " the great misfortune of the peasants is to be foolish (*imbécilles*), and another is to be too much neglected: no thought is given to them except when a pestilence desolates them, themselves and their herds . . ." The greatest pestilence desolating the herds of Western Europe towards 1851 was pleuro-pneumonia which, after the lifting of restrictions upon importation of live cattle from the Continent, reached England in 1841, foot-and-mouth disease having arrived two years earlier, in 1839. Cattle plague was not to reach our shores till much later, in 1865, but then agriculture had not reached the end of its " Golden Age " of prosperity and the country could well withstand the great costs of eradication, which in its train brought about nearly the complete extinction, too, of pleuro-pneumonia and, temporarily, of foot-and-mouth disease. And, so, that costly outbreak did not move the Government to make further provision for veterinary education, even though the Cattle Plague legislation of the period laid the foundations of the present-day Animal Health Division of the Ministry of Agriculture.

The somewhat harsh qualification, wrote Bouley, applied by Voltaire towards the end of this last letter to the peasants of his time would appear to merit application to some of our own time. [Voltaire wrote in 1775, i.e., 68 years earlier than Bouley's comment, in 1853.] The following anecdote furnishes proof. We extract it from the memoir on contagious bovine pleuro-pneumonia which M. Huvelier, a veterinarian practising at Alençon, submitted to the *Société Centrale Vétérinaire* for the prize competition held in 1847 . . .

Subterfuges employed to exorcise a supposed spell cast upon cattle that were dying of pleuro-pneumonia.

Two residents in the commune of Belfonds, who had formerly been friends, became enemies through each coveting what the other desired. One of them, B——, who for a long time had taken to drink, had, it was alleged, uttered threats against Mr. H——. At the time of the pleuro-penumonia invasion on the premises of the latter, these threats, which had been unheeded when first they were uttered, came back more vividly to memory on seeing that the disease continued its ravages for two whole years. It was concluded from this circumstance at H——'s place that it was his enemy B——, by his evil designs or through people hired by him, who was causing the death toll, till then unnoticed, among the stricken cattle.

To try to bring this calamity to an end, H—— proceeded to Caen to a sorcerer of great renown so as to cause the spell to be lifted which was ruining him. The sorcerer, in his greed for money, missed no chance of entertaining H——'s suspicions and affirmed that he would take it upon himself to trace the guilty party and punish him by the law of retribution. Unfortunately, appearances pronounced themselves all in favour of this impostor, whose infamous conduct should have been denounced to the competent authorities.

This is what happened afterwards:—

The great sorcerer repaired to the premises of Mr. H——, shut himself up in the cattle sheds for a long time where he practised a number of ridiculous mummeries accompanied by lugubrious howling; there was burning of sulphur

and several parodies of religious practices. Then, when the spirits were well roused, he gave orders that a calf be brought to him, whereupon he cut its throat and suspended the agonised brute from the ceiling, thrusting into the heart from the first day a large nail and declaring that if the guilty party were someone in the commune he would not have to wait long before he felt the most violent pains in the chest with each succeeding nail thrust into the heart after the first one. It goes without saying that this abominable man had drawn from Mr. H—— all that he wished to know relating to the suspicions which he entertained against Mr. B——.

To make easy an understanding of the facts it is not without point to recall that the latter had become demoralised for a long time by drink as well as from chest trouble and was beginning to show a dropsy that was not a secret to anyone. Public clamour denoting him deafeningly as a caster of spells, he fled from all his neighbours who would have sooner given their hand to a murderer than to a man who was even suspected of having poisoned or bewitched a few cattle. Kept informed of what was going on and not daring to face the storm by addressing a complaint to the authorities, B—— was overtaken with such terror that he made an attempt at reconciliation with H——, which the latter rejected on the advice of the sorcerer. Moreover, to wish to become reconciled in such circumstances, was it not showing that one feared discovery of the truth? Was it not an avowal of guilt? Thus, this step succeeded in ruining B—— in the opinion of the commune.

I have been assured that, during this interview, in rejecting the peaceful overtures of B——, H—— foretold to him that he would soon die of the same disease as the cattle. The unfortunate man became so terrified that the very next day he went to bed never to rise from it again.

It is said that in his delirium he called out to H——, with loud shouts, asking him pardon and confessing himself guilty of the death of his beasts. However, a fresh nail was driven each day into the heart of the hanging calf, until it ended by all nails falling out when that organ had become putrid, the time limit assigned for the punishment of the author of the misfortune. Kept in touch with the course of events by indiscreet talkers, B—— was no longer in a state of life and had only a few lucid moments, his sufferings becoming visibly worse. The sorcerer, who was being acquainted with everything and to whom the turn of events exactly suited his wishes, suddenly announced that he was going to attempt the final proof and that the name of the guilty party at last was going to be divulged. For this purpose two close relatives were ordered to revolve a winnowing van into which were to be thrown several rolled-up tickets, and it would be on the ticket which the van on turning hurled out farthest that would be revealed the detestable name! What was said was carried out; the ticket hurled farthest, as we might well expect, bore the name of B——. It was therefore he, without question any longer, who had caused the cattle of H—— to perish.

Informed of what had happened, B—— was so much stricken down that he died almost at once; the public, instead of attributing his dropsy to the abuse

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of strong drink, found it quite natural that he had been punished for the evils of which he was the perpetrator. Thus were realised the foretellings of the sorcerer whose renown in measure grew greater.

How can you expect from now onwards, for a long time to come, people in the commune of Belfonds not to believe in sorcerers!

All this happened in the Department of Orne, in the year 1846.

After the death of B——, all the cattle of H—— being dead or sold, the disease disappeared, like a fight, for want of fighters (*comme le combat, faute de combattants*).

H—— had the good sense to wait until the spring before buying fresh animals, which remained exempt from pleuro-pneumonia, to the greatest glory of the sorcerer who had lifted the spell by immolating the one who was casting it.

VIII

We would have not lightly trespassed upon the space of this Journal by reproducing what will seem to most readers at this day to be an idle anecdote. But in the year 1851 the prevailing mood of the countryside, nurtured as it was upon the superstitious and hysterical in religion, the mystical, and the supernatural, was genuinely emotionally susceptible to the reception of impositions of the kind vividly recorded above by Bouley. If it did nothing else, rudimentary again as it may seem to us judged by present-day standards, the veterinary education of the time imparted at the schools at least freed the countryside from the toll exacted from it by hosts of predatory impostors preying upon the credulity of the stockowners. Much documentation could be reproduced, did space permit, to support the view that conditions were not very much better in Britain at that time than they were in France.

IX

The most recent historical monograph to hand [Briggs, A. (1951), " 1851," Historical Association, G18] contains the following apposite passages we make bold here to reproduce: "... *The Exhibition, the political crisis, the religious hysteria, all contributed to the temper of* 1851 (our italics)... The Exhibition showed the nation's undisputed leadership in manufacturing. Its engines were the symbols of economic mastery... In commerce and finance, British supremacy was even more marked. Yet despite spectacular signs of progress in trade and industry, over a quarter of the male population of the country was engaged in agricultural pursuits... 1851 was the critical year in the reshaping of British agricultural prospects. It ushered in a period of agricultural prosperity, largely dependent upon the opening of the railway system and the increase in incomes generated by industry... There was... an underlying distrust of government, which continued to mould the shape of politics until the passing of the Second Reform Bill. Self-help came first: *government, except as an agency for dealing with foreign relations and for facilitating self-help, came very low on the list of daily pre-occupations... It certainly came below religion.* (our italics) Religious questions shared the headlines with news of the Exhibition throughout the first

half of 1851." And then at the conclusion of this learned survey the author states, quoting Professor Woodward, " the Victorians were living dangerously, far more dangerously than they knew. The world was much stronger than their machinery, and the nature of man more fragile and at the same time more unfathomable. "... Men are beginning... to feel increasingly nostalgic about the loss of the world which reached its triumphant peak in the year of the Great Exhibition."

X

We can now survey the scene for which, despite all that has been said, we in Britain need suffer no shame in so far as it conveys a disinterested impression of the British people of the time, as seen through the eyes of a sympathetic foreign colleague, M. Gourdon, from the Veterinary School at Toulouse, France. That scene was portrayed in the *Journal des Vétérinaires* (1851, Nos. 7, 8 and 9), from which Wm. Percivall published the following excerpts in the *Veterinarian* (1852, *25*, 201):—

Landed at Southampton, M. Gourdon finds himself in a town whose streets, running at right-angles, are large and gloomy and silent, in the midst of misty, penetrating rain, with a grey sky over his head: unvarying specimens, he adds, of the atmosphere of Britain.

From Southampton he proceeds by rail to London, and in the course of his journey is struck with the use made of every particle of land for the purpose of agriculture. All is in a state of cultivation. Inclosure succeeds inclosure without interruption.* Here are cornfields, meadows and pastures; further on are square plots of garden ground, all neat and clean and regular; drilled in lines, weeded with care: in a word, tended to with a solicitude denotive of an advanced state of agriculture. Nor do we see land uselessly encumbered with large hedges; but, instead, partitioned off by railings or fences of wood, evincing the care the English take of losing nothing through a want of reciprocal good feeling between the several proprietors of adjoining lands.

He arrives in London, the immense commercial bazaar of the world; the town of barter and business, wherein pleasure becomes the exception, and life without business is despised; the town of contrast, wherein the most colossal fortunes rub elbows against the most squalid wretchedness; certain parts of which display scenes of great agitation, while in others nought is to be seen but cold and tristful solicitude; the town of fog, of rain, and of mud, whose styptic climate freezes the imagination and destroys the energy of thought, leaving to man nothing save the faculty of the enjoyment of things to the extent of their absolute utility, yet a faculty cultivated by the English to the highest point with every conceivable ingenuity!

But M. Gourdon, on better acquaintance with it, finds London—by way of compensation as it were for the *desagremens* he at first encountered—an exceptional city, inasmuch as everybody in it enjoys full liberty of movement,

*Enclosure of all commonable land in England had only just been completed at the date of the Great Exhibition of 1851.

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act, and word, and is as free as though he were in the midst of a desert; wherein, indeed, a foreigner, and especially a Frenchman, finds himself after a four-and-twenty hours' sojourn, instinctively relieved of his national individuality, which he in vain endeavours to reassume; in fact, a free town, open to everybody, without any annoyance of police, without hypocrisy, and with full liberty of thought.

"With this preliminary effusion, let me," continues M. Gourdon, "commence my undertaking. But where shall I make a beginning?—and of what shall I speak first? How am I to make an election, humble as I am, and uninformed about so many things, in the midst of such an immense assemblage of industry and talent from far and from near, combining specimens of all kinds of art and science in their newest forms, with *chef d'œuvres* unknown to the world, and perfectionateness unexampled? Even up to the present hour, while holding my pen in my hand, I feel myself posed by this question, and dazzled and confused for the second time, the same as I was at my first entry into the palace in Hyde Park: a sensation I continued to experience for several days afterwards."

At length, M. Gourdon makes a beginning in the "anatomical department," when he proceeds to the examination of the *maréchalerie,* or farriery department.

"This occupied a corner of the Exhibition little frequented by the pressing and elegant crowd of persons present; though for my own part, I profited by the occasion to attentively examine what was deposited there, with the view of learning if it contained anything that was really new. In the collection I counted 129 horseshoes, all of English make, distributed into eight collections, every one more or less resembling another. The shoes, symmetrically arranged and highly polished as they were, looked well enough to the eye; and this is pretty well all I can say in favour of them; since, in a practical point of view, I saw nothing, after a long and tedious search, worth the pains of giving an account of to French veterinarians and farriers.

"All, or nearly all, possessed the fullering so characteristic of English shoeing, as well as the bevelling off of the inner border of the web for the sake of adaptation; and most of them were exhibited as perfections to prevent slipping.

"With this view, imagination had been stretched to its utmost to stamp the most fantastical irregularities upon the exterior of the ground surface." M. Gourdon then gives an illuminating detailed description of the several types of horse shoes on view (which the interested reader can consult in the original), giving it as his conviction that the French system of shoeing, by merely leaving the heads of the nails projecting out of their holes was far superior to the innumerable modifications made to the simple horse shoes to prevent slipping or foot expansion. He concludes: "... and yet the English, or the majority of them at least, refuse to entertain it, under the pretext that, though our plan of shoeing may answer very well for us, it is worth nothing to them; at least, such was Mr. Field's answer to my enquiries on the subject, without, as I thought, assigning any satisfactory reason: showing, when all comes to be reckoned up,

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that this amounts to neither more nor less than purely a piece of that national pride to which the English are so prone, and especially when any comparison with us French, to whom they will on no account confess inferiority, becomes the question at issue. But judgment is declared. On this, as on many other questions which the Exhibition has cleared up, France has carried off the prize without contest; not in the Crystal Palace, to which she did not send so much as the head of a horsenail, but at home, within the improved forges of her principal towns, wherein scientific farriery, aided by manual skill, has become a finished art. And if there be, at the present day, any horse proprietors, any '*gentleman riders,*' or English veterinarians, who refuse their belief to evidence so clear and which is substantiated by several of their countrymen, let them avow the only truthful reason for refusing to subscribe to that in opposition to which the best interest of horses, viz., the healthy practice of our art, absolutely can discover nothing to urge."

XI

The remainder of Percivall's editorial (*Veterinarian*, 1851, *24*, 596), to which we alluded at the beginning of the present article, exhausts the literature available, so far as we know, bearing upon the Great Exhibition and in so far as it supplements M. Gourdon's narrative, and lends confirmation to the main thesis or the article, it merits reproduction:—

". . . The professions, it is true, as contra-distinguished from the trades, are not of a nature to admit of their science being displayed to much advantage in a public show; though the branch of medicine we call *surgery*, from the hand being the agent through which, with the aid of instruments and appliances of various kinds, operations are performed of vital service to mankind, creates an interest in the arts which the public, as well from curiosity as from sympathy, evince their encouragement of it. People are curious to see the instruments used in amputating limbs, and extracting stones, and trephining, etc.; they likewise look with interest on the productions, natural and chemical, out of which the doctor culls remedies for those ills which so easily beset them. In veterinary surgery, displays may be made of cauterising and other instruments, as well as of apparatus used for casting and securing horses and other animals in order to render them subservient to operations deemed necessary for the removal of their infirmities or deformities. But, in the veterinary line, few things are apt to create so great an interest in the public mind as *horseshoes*, of which some of the best make of our own, and some of rougher manufacture, American, appear in the Exhibition. Both in instruments and horseshoes, indeed, something has been done for veterinary science, but it has been very moderately done. The instruments are but few, and the horseshoes are but old acquaintances with brightened faces or in handsome cases.

"There is, however, an article on exhibition in the French department which may well lay claim to our attention. If not quite new in its design, it is at least of novel character with us, and will, if we are not mistaken in our estimate of the merits of the ingenious fabric, elicit—if it have not already

elicited—from veterinarians greater admiration than anything in their province which the Crystal Palace contains. A Dr. Auzoux has brought with him from Paris, for exhibition, wax models of the human and equine bodies, so constructed that, like a child's puzzle, admitting of being taken apart, piece by piece, they display to the spectator."

Percivall then proceeds to discourse at length on these wax models, their probable utility, and the need to raise funds to purchase them for the Royal Veterinary College.

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At this date, all that seems trivial, even tedious, as doubtless much of the same *genre* of exhibits will appear to our descendants a hundred years hence in the records left of the present exhibition.

After all, the craft of "surgery" in 1851 had scarcely been separated by longer than a few decades from that of the barber, and both branches of the original barber-surgeon craft, to the eyes of a scientific research worker, have indeed progressed so admirably in the art of exhibiting themselves that an up-to-date flourishing hairdressing establishment bears a curious resemblance to the surgery premises of an efficient and successful small animal practitioner.

Whether that is the *summum bonum* of veterinary achievement we can leave without comment, having reported, conscientiously, we hope, at the beginning of the present article wherein the State and the public have now beckoned to the veterinary profession lies henceforward its solemn DUTY.

(To be continued)

GENERAL ARTICLES

NECROTISING ENCEPHALOMYELITIS (or Encephalomyelomalacia) of unknown Ætiology in Goats in Ceylon with similarities to Setariasis of Sheep, Horses, and Goats in Japan

(Including a brief review of nervous diseases of goats)

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Introduction

THIS paper emanated from observations of Crawford (1939-42) on a disease of goats in Ceylon known as " lumbar paralysis." Comments on the clinical features and speculation on causation were all made by Crawford, but the exact nature of the condition eluded identification until now and is dealt with in this study. The literature on nervous diseases of goats in general is reviewed. The limited material available for pathological study might restrict the value of the observations; however, the nature and selective distribution of the lesions indicate that the pathology of one paralytic syndrome in goats in Ceylon has been established.

There is little known about nervous diseases of goats: all the references are given by Innes (1950), but a few relevant ones are cited. Subsequent to the work of Gill (1931) in New Zealand on the rôle of *Listerella Erysipelothrix monocytogenes* in " circling disease " of sheep, comparable infections were found in the same, and other, species throughout the world (see Graham, *et al.* [1943-4]). When the nervous system was involved the process has been a purulent meningo-encephalitis, meningeal œdema and purulent foci in nervous substance with perivascular " cuffing " affecting mainly the brain stem, hippocampus and corpus striatum. From the descriptions (and cases in sheep seen personally) there is necrosis virtually of all tissue around the organisms. King (1940) described an outbreak of listerellosis in goats (U.S.A.) as a disseminated encephalomyelitis with granulomatous lesions almost restricted to the brain stem; the changes were regarded as a variety of myelin loss to which later reference is made. (Papers by Olafson [1940], Gifford and Eveleth [1942], and Kaplan and Lager [1945] deal with isolated cases of listerellosis in goats in the U.S.A.) The above facts are mentioned, for it must be stressed that no encephalomyelitis due to *Listerella* has been observed in *goats* in England, Ceylon or Japan. The records of Burns (1944), Pavlov (1940) (tick fever), and Verhaart (1942) (cerebellar agenesis) on goat diseases do not suggest any clinical, or pathological, similarity to this " lumbar paralysis " in Ceylon. There is no further mention of caprine nervous diseases in Nieberle and Cohrs (1949), in Scherer (1944) (apart from that to Emoto's and King's papers), in Hutyra, Marek and Manninger

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(1946), or in the "Handbook on Diseases of Goats," N.V.M.A. (1950). The above references nearly complete the published studies made on any caprine nervous disorder in Europe and America; the congenital myotonia (Thomsen's disease) of goats described by Kolb (1938) and Brown and Harvey (1939) is an unusual, totally different disease to lumbar paralysis. *Krimpsiekte* of goats, said to be caused by poisoning with *Cotyledon wallichii* in South Africa, produces spasms and other nervous signs, but without lesions in the nervous system (see Hennings, 1932, and Steyn, 1934).

Emoto (1927) studied a paralysis of goats in Japan which occurred from 1911-24, all the animals having been imported from Switzerland. (The editor of the journal in which the article appeared commented that the condition was unknown in Switzerland.) The disease was seen each year in the late summer and autumn; adult animals were usually affected and became totally helpless, paralysed in the hind limbs or showed mild incoordination; the course was mostly acute, and death often occurred from pneumonia. Emoto designated the condition a non-purulent lymphocytic meningitis with cerebrospinal malacia, the lesions of which showed no hæmorrhage or neuronophagia, but consisted of vacuolar and chromatolytic degeneration of neurones, inflammatory cell infiltrations and degeneration in the cord. Unfortunately, the descriptions are in general terms and unillustrated, so that the condition of the myelin or of the axis cylinders and the distribution of the "cord degeneration" are ill-defined. Sometimes only segments of the cord seem to have been affected, and this might have accounted for the variety of severity of the "pareses." The claims that a streptococcus was the causal organism might be regarded sceptically; no positive results followed injection of the streptococci by subcutaneous or visceral routes, while goats inoculated intracerebrally were alive after a few weeks. The descriptions of the lesions, further, might not harmonise with those expected in any streptococcal infection. Hashizume (1940) confirmed many of these observations of Emoto, but his cultural and experimental work did not establish any bacterial cause.

General Observations on Lumbar Paralysis in Goats in Ceylon

The disease attracted attention in Ceylon from 1931 onwards, following importation of goats from India, and comments on certain features were made by Crawford (1939-42). Although there were no other references found then in the literature dealing with such a disease, caprine paralysis had been mentioned in the Annual Reports of the Muktesar Laboratory, India, between 1934-42. In 1934 (p. 31) it was stated that a paraplegic goat showed lesions of acute encephalomyelitis, and in 1940 (p. 28) that it was an obscure disease in the Bhadia Farm, United Province, and that attempts to transmit the condition were unsuccessful. The disease was "clinically characterised by nervous symptoms indicating an encephalomyelitis, and the cerebral cortex of affected animals showed lesions specific for the disease—meningeal congestion and hæmorrhage, perivascular infiltrations, neuronophagia and destruction of nerve cells and similar lesions were found in the cord" (p. 35). In 1941 (pp. 7 and 21) a severe

PLATE I

FIG. 1
Spinal cord, lumbar paralysis, Goat, Case 8. Showing diffuse involvement of ventro-lateral columns of white matter with spongy state; cracks and crevices; prominent vessels. H.E. x 20.

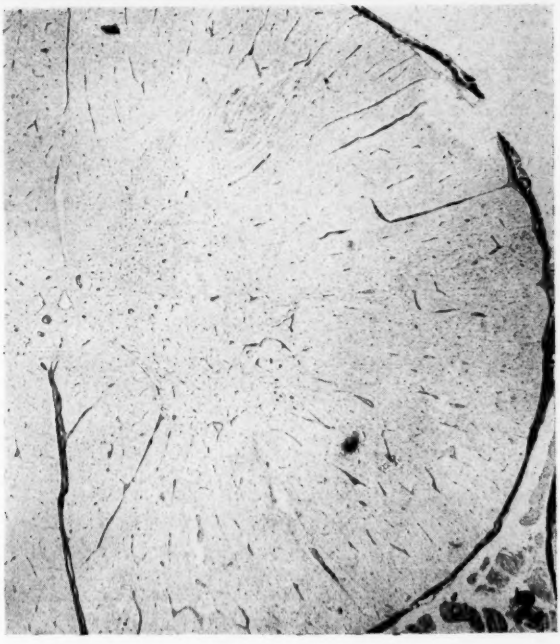

FIG. 2
Spinal cord, same case as Fig. 1 but different level; reticulum stain (Wilder) showing prominence of radially disposed vessels due to increase of argyrophil fibrils. x 20.

(*Article by Innes, page* 187)

PLATE II

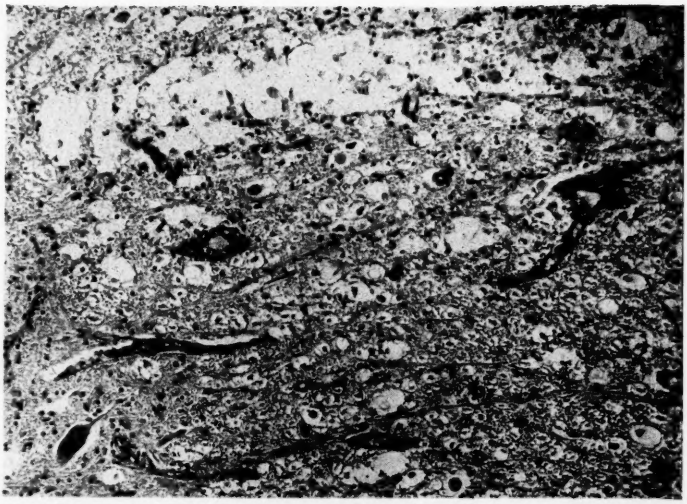

FIG. 3
Spinal cord, same case as Fig. 1. Showing spongy state, part of which has caused a "falling out" in white matter adjacent to ventral horn of grey matter (extreme bottom left); all vessels show cuffing; ballooning of myelin sheaths; swellings of axis cylinders seen as homogeneous round blobs in spaces. H.E. x 90.

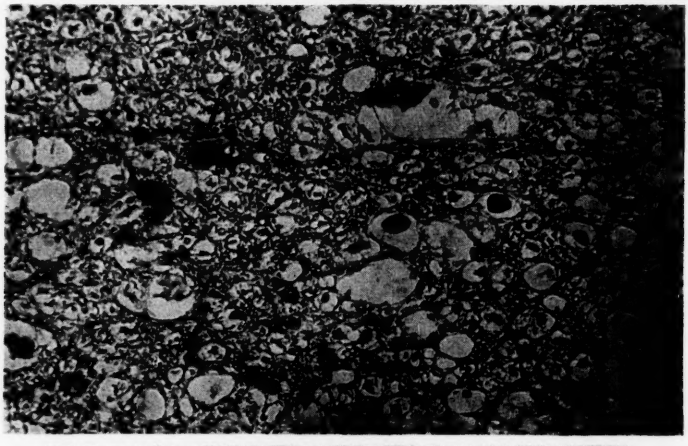

FIG. 4
Spinal cord (Case 4). Showing many round empty spaces which probably contained swollen axis cylinders which have fallen out; gigantic swelling of axis cylinder at bottom right. (arrows) x 90.

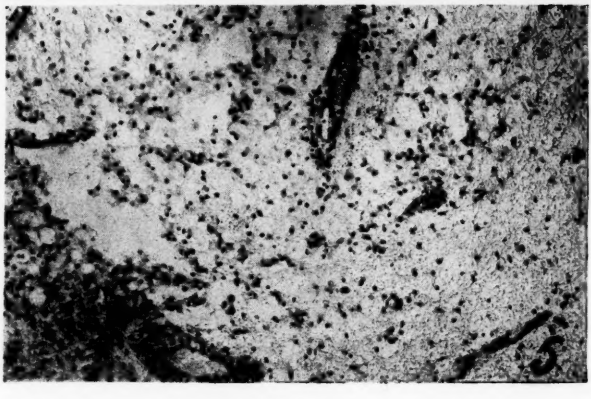

FIG. 5
Spinal cord, another field from the same case as Fig. 3 stained by Nissl. Cuffing of vessels and proliferation of astrocytes in softened area. x 90.

(*Article by Innes, page* 187)

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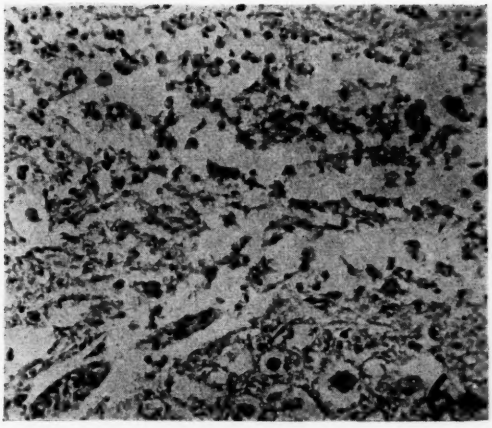


FIG. 6a
Spinal cord. Same case as Fig. 4. Focus of very advanced tissue dissolution numerous compound granular corpuscles ("gemästete glia"). Weigert's hæmatoxylin. x 90.

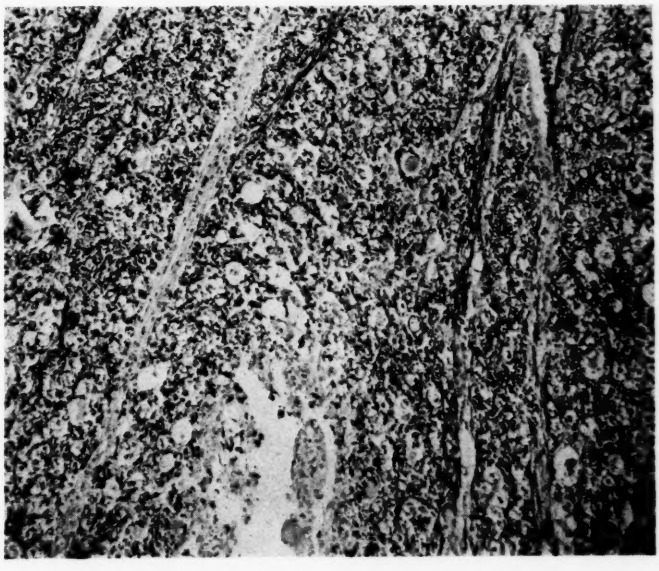


FIG. 6b
Same case as 6a. Myelin stain on paraffin section: showing complete disappearance of myelin in parts: swollen axis cylinders have not retained hæmotoxylin. Modified Loyez. x 90.

(*Article by Innes, page* 187)

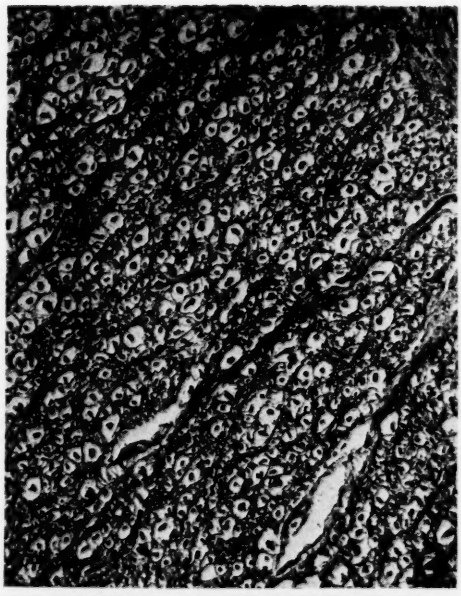
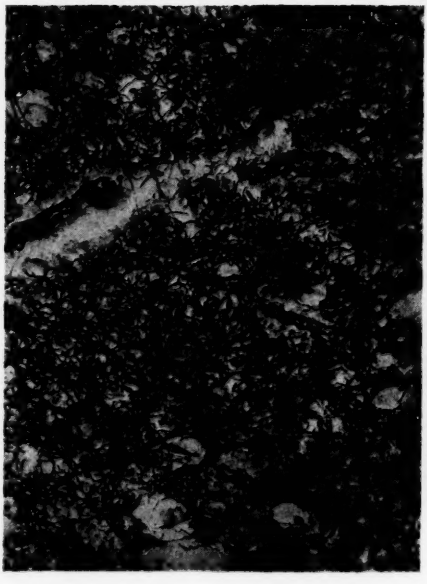


FIG. 14
Spinal cord, normal control goat. Transverse section through tip of ventral horn of grey, and ventro-lateral column of white, matter. Showing disposition of glial fibrils after Hortega's silver carbonate method, frozen section; compare with Figs. 15 and 16. x 185.

FIG. 15
Spinal cord, same case as Case 8. Showing thickened vessels and some gliosis at edge of lesion where there was little to be seen in Nissl or H.E. except a few swollen axis cylinders (arrows). x 185.



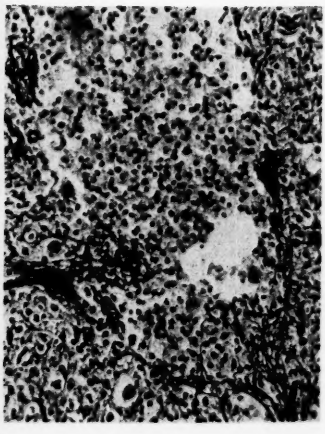


FIG. 16
Spinal cord, same case as Fig. 15. Showing intense gliosis in midst of softened area. Hortega silver carbonate and frozen section. x 185.

FIG. 17
Spinal cord, same case as Fig 16. Showing a large clump of "*gemästete glia*" which have held together, surrounded by impregnated fibrils some of which are glia but also reticulum related to vessels; a few shadows of swollen axis cylinders. Cajal gold chloride - sublimate, frozen section. x 300.

(*Article by Innes, page* 187)

outbreak of encephalomyelitis in cattle was reported to have caused many deaths and a few cases were detected in goats, but sporadic cases had occurred since 1931. The ætiology of this condition in goats in India was not known and it is unfortunate that more descriptive illustrated data of the lesions are unavailable which would help to distinguish its pathology.

The main signs of the disease (Crawford, 1939-42) confirmed by Malkani and Pillai (1950) were motor-weakness and posterior incoordination. In some goats there was swaying and stumbling, while in more severe cases there was complete loss of motor power in the hind limbs or all four legs. The onset was sometimes dramatically sudden, and a goat might be found lying on its side unable to rise; some might show nystagmus; the disease was non-febrile. Some animals died in a few days, but many survived and then might show some clinical improvement but with impaired limb mobility; in yet other cases the onset was insidious and the first sign was simple unsteadiness of the hind limbs which might not be progressive. Other information of diagnostic importance which could be obtained by attention to neurological methods is unavailable. This " paralytic " syndrome might cover distinct pathological entities which can be elucidated by more study, for in the same way " paralysis " of lambs in England and Australia was first a confusing clinical hotch-potch before pathological clarification.

The cases seen years ago by Crawford were confined to imported goats or goats bred from imported stock: on some farms to which imported goats were sent, and on which cases occurred, the local breeds did not suffer. The disease had no epizootic trend, being mostly irregularly sporadic, or only isolated cases were noticed at long intervals or minor " outbreaks " involved only a few goats.

The disease has now been seen by Malkani and Pillai (1950) in a variety of Indian breeds imported into Ceylon—Jamnaparais, Scindis, Kamoris, Alandis, Bangaloris, and in crosses between these and local Ceylon goats; two cases were seen by Crawford in 1938 in a dealer's yard in Karachi (India), but these were not locally-bred Indian animals.

The fact that the disease (see also reference to Emoto) was confined to imported stock engendered an idea that it was some infection to which indigenous stock were resistant. Unsuccessful attempts were made by Crawford to transmit the disease by inoculation (subcutaneous and intramuscular) into healthy local goats of brain and cord emulsions. He found it impossible to produce any infection in rabbits, guinea-pigs or mice by the same tissue emulsions, while aerobic cultures of blood, spinal cord and brain, using ordinary media, showed no growth of pathogenic organisms. The history of the disease did not suggest a high degree of contagion or infectivity. The well-known virus encephalitides (except rabies) of other animals have not been identified in Ceylon, and the same paralytic syndrome has never been encountered in other native domesticated animals in Ceylon. Rabies is common in Ceylon (dogs, man, horses, cattle, pigs, and goats), but can have no clinical or patholgical resemblance to " lumbar paralysis."

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The nervous system showed no macroscopic changes (Crawford). In some preparations of the spinal cord (Marchi's technique) there appeared to be a "demyelination," as evinced by the reduction of the osmium tetroxide, but this was doubtful. Without more exact knowledge of pathology, efforts to elucidate ætiology could only be tentatively undertaken. The clinical syndrome, although like that described in Indian goats (Muktesar Reports) and in the goats in Japan (Emoto and Hashezumi), might involve different diseases. Crawford stated that spinal injury, for example, sometimes occurred in male goats required to serve small females, and milk fever in female goats might be mistaken for "lumbar paralysis." He thought there was some superficial resemblance between caprine paralysis and lathyrism in man and animals, and it was found that many samples of gram (*Cicer arietinum*) fed to stock goats were contaminated with *Lathyrus sativus,* but the cessation of feeding such gram did not prevent the disease. A young goat (local breed) was fed for a long period on large quantities of *Lathyrus sativus* without causing any nervous symptoms. The possibility of poisonous plants being involved inspired Crawford to make other feeding experiments, but with negative results; the plants fed included *Smilax zeylandica, Epaltes divoriticus, Calotropis gigantica, Pavatta spp.,* and *Lantana camara.* The probability of tick paralysis being involved was contemplated by Crawford but never settled. Data were unobtainable of the pedigrees of the affected goats, but the occurrence in such a variety of breeds from widely separated parts of India precluded any concept that it might be an inherited disorder. Only adult animals were affected, and some of those had normal progeny.

The condition might bear comparison with "kumri" of horses in India and Burma, a disease also characterised clinically by posterior incoordination. From the meagre descriptions of the pathology (see Rippon [1941]), this disorder (encephalitis) might be comparable, although this has not been elucidated. It was unlikely that there was any real relationship, for there are few horses in Ceylon and the chance of contact with goats would be remote, unless, as Crawford postulated, both horses and goats became "infected" from a common reservoir such as some arthropod vector (see addendum).

Pathology

Material and Methods.

The brains from eight cases (spinal cords from six of these) of "lumbar paralysis" in goats were available for examination. Autopsies were carried out,* and the nervous system fixed in 10 per cent formalin saline before dispatch. Unfortunately, in most, fixation was not good; consequent to this and packing, some of the tissue suffered distortion and in parts showed much post-mortem change. This did not exclude study of many important features of the lesions, although some qualifications might be attached to changes in certain structural elements. Coronal sections were cut, 2-4 mm. thick, starting at the frontal poles and working back; when the cord was available sections were made at

*By Mr. C. Perumal Pillai, Veterinary Laboratory, Peradeniya, Ceylon.

many levels. Sections were prepared after paraffin embedding and after the frozen technique; routine stains were used, as well as many selective neurological techniques on both paraffin and frozen sections for the study of neurones, myelin, axis cylinders, neuroglia, and lipoid. There were no apparent macroscopic changes in the fixed material sent.

Protocols.

Case 1—(Goat No. 14). Karadian Aru Farm, Eastern Province. Typical symptoms of lumbar paralysis on 8.6.49, died on 21.6.49. Only brain sent. (N.I.H. 47290.)

Case 2—(Goat No. 5). Karadian Aru Farm. Born 23.2.48, Jamnapari breed. Inco-ordination of hind quarters, affected with parasitic enteritis and coccidiosis, died on 10.2.49. (N.I.H. 47289.)

Case 3—(Goat No. 16). Jamnapari, imported Indian breed. Four or five years, male. Moderate swaying of hind quarters in August, no improvement in 3 months, and animal killed. (N.I.H. 47715.)

Case 4—(Goat No. 17). Jamnapari. Four to five years, female. Normal parturition 2 months before onset of paralysis; unable to rise; duration of paralysis about 2 weeks. (N.I.H. 47716.)

Case 5—(Goat No. 13). Jamnapari. Eight months, male. Duration of paralysis about 5 months. (N.I.H. 47754.)

Case 6—(Goat No. 15a). Jamnapari, Indian imported. Male, 4-5 years. Duration of paralysis unknown. (N.I.H. 47755.)

Case 7—(Goat No. 18). Jamnapari. Female. Onset of paralysis 6 days before normal parturition, death $3\frac{1}{2}$ weeks later. (N.I.H. 48250.)

Case 8—(Goat No. 19). Jamnapari. Female. Normal parturition 3 months before paralysis. Duration of paralysis 6 days before being killed. (N.I.H. 48251.)

Histology.

In four of the eight goats, examination of the nervous system revealed no lesions (Cases 3, 5, 6 and 7). The histological characteristics of this caprine disorder, as described below, are unmistakable even in sections prepared by routine stains. A large number of slides were made from the brain and cord of these four animals and the possibility that a lesion was missed is thus remote.

In the other 4 cases* there were scattered cellular infiltrations in the cerebrospinal meninges (lymphocytes, polymorphs, eosinophils). There were no hæmorrhages in any part of the nervous system, little vascular dilatation and no thrombi. The ventricles were not dilated and the ependymal cells were unaltered except for desquamation due to inadequate fixation. Search for inclusion bodies in the cytoplasm, or nuclei, of neurones, glia, vascular endothelium, or ependymal cells was negative. In preparations stained by Gram's method there were no Gram-positive bacteria, while other specific stains did not demonstrate parasites such as toxoplasma, encephalitozoon or sarcosporidia. In these four cases in which

* Another identical case has been studied since.

lesions were found, there were none in the grey, or white, matter of the cerebral hemispheres, and certainly none of the severity found elsewhere in the brain stem. There were lesions affecting almost predominantly the white matter of the latter and not all at the same segmental level. In one goat there were focal asymmetrical patches in the mid-brain as well as cord; in another the cerebellar white matter and pons were involved, but, unfortunately, in this case there was little spinal cord available. In the remaining two, lesions were present only in the cord and were prominently distributed unilaterally in the white matter; equally striking was the fact that the changes were confined in longitudinal magnitude. Hence, random selection of a few transverse slices of cord for examination might have resulted in a lesion, or multiple foci, being missed, for macroscopic inspection of pallid fixed material was no help in determining damage. From a consideration of the nature of the lesion it is possible that careful naked-eye observation of fresh tissue might give a different answer. In one of these cases transverse sections were made at fifteen different segments and longitudinal preparations of many portions between; a single lesion was detected in one of the transverse cuts made about C.6, and from the way it faded out above and below the focus might have been about 2 cm. long (also seen in a 1951 case). This gives some indication of the examination which might require to be made in suspected clinical cases.

In H.E. and Nissl preparations of a lesion in the cord (Figs. 1 and 3) the white matter was preponderantly involved, and in a transverse plane almost the whole of the ventrolateral column of the white matter and the commissure on one side was implicated diffusely, yet irregularly, and not confined to distinct tracts. The motor (e.g., rubrospinal) and ascending spinocerebellar tracts were all affected. In only one case were minute lesions found in the dorsal tracts. In the implicated segment the white matter appeared almost like that of a *status spongiosus* with a disorderly array of cracks and crevices (Figs. 1 and 2). In the more severely affected parts the whole ground substance must have been loosened and destroyed. Some allowance must be made for this spongy state to have been artificially exaggerated by a "falling-out" in sections of grossly malacic zones. This was apparent in frozen and paraffin sections and was a factor impeding preparation of intact sections. Better fixation immediately after death might have improved this defect. Almost all the vessels in the lesion possessed extra-adventitial cuffs, or sleeves in those vessels disposed radially; the cells in these infiltrations were lymphocytes, mononuclears, and occasionally eosinophils and polymorphs. *Gemästete Glia* were moderately numerous around some vessels or even in the cuffs, but they were more prone to form clumps; many of the crevices must have been packed with the same cells, and it appeared to be these foamy cells which tended to drop out most. These glia were more prominent in areas where white matter destruction had been most marked. Nissl preparations showed augmentation of cells with large, clear, vesicular nuclei (astrocytes) in the white matter as compared with the numbers in the contralateral unaffected columns (Fig. 5). Myelin stains (frozen and paraffin) demonstrated loss of myelin in fibres around vessels, but also in a more

moth-eaten way in the spongy areas (Figs. 6b and 10); there was no proclivity for this myelin loss to be systematised and to affect tracts. An impression was that the fibres were more seriously destroyed from the pia mater inwards; even so, through the ventrolateral column there were still many fibres which still retained the myelin stain; ballooning and fragmentation of involved fibres was evident under higher magnifications. The *gemästete Glia* seen as foamy cells in H.E. and Nissl contained fine droplets of lipoid-staining material in frozen sections (stained by oil red-o), and this was also apparent by Marchi's technique. This demonstrable lipoid was never abundant nor such an outstanding feature as in some demyelinating lesions of man and animals. In cuts of the cord made above and below the lesion, stained by Marchi, there was indisputable secondary degeneration (c.f. changes in axis cylinders), but many of the black granules in the myelin sheaths might have been artefact. Hæmorrhage was absent in lesions, but occasionally minute perivascular red cells were found in the grey matter.

A salient feature constantly seen in all lesions, to which attention was immediately drawn, concerned the axis cylinders. In H.E. and Nissl stains (or, indeed, any stain—Van Gieson, methyl violet, Weigert's hæmatoxylin, gallocyanin, phospho-tungstic acid) irregular globose bodies were scattered throughout the malacic area (Figs. 3, 4, 6a and 13); these were homogeneously stained pale by cytoplasmic stains or were faintly granular. They lay in the centre of what was ostensibly a tube cut transversely—representing the myelin sheath; it was evident they tended to fall out, and perhaps many of the roundish spaces seen in the paraffin sections had contained such bodies (Figs. 1, 3 and 4). They were unstained by Marchi and lipoid dyes, but had an affinity for the silver of the Bielschowsky method (or various modifications); they were stained in a similar way with other silver impregnation methods (e.g., Wilder for reticulum, or Hortega for glia) and were well demonstrated by Cajal's gold chloride-sublimate method (Fig. 13). They varied enormously in size; some of the smaller were 2-3 times the transverse diameter of normal axis cylinders; others were gigantic, and perhaps 30 times that of the normal. Their position and their affinity for silver denoted that these changes represented some abnormal response of the axis cylinders. This had not resulted in complete disintegration along with that of the myelin, but in peculiar torpedo swellings, corkscrew spirals, globular balls, and, in fact, a bizarre array of shapes and sizes. Some might have been terminal, glomerular-like balls, others collateral excrescences of axis cylinders, and there were also varicose beadings (rosary effect) along their course. This was clear in longitudinal sections or if the fibres were seen obliquely (Fig. 12). The same type of change in axis cylinders was sometimes seen in the spinal nerve roots. All those features, together with the absence of reaction of these " bodies " for stains specific for hyaline substances, indicated that these changes in the axis cylinders were characteristic for whatever form of inflammation existed in the nervous system of these goats. The same axonal changes, but to a less degree, were also found in these lesions which were found at higher levels in the nervous system of two goats. (The initial impression that they might have been myelin balls, amylaceous concretions or some queer,

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foreign substance was thus erroneous.) Large, swollen axis cylinders were found in zones where myelin had entirely disappeared. All swellings were granular or intensely impregnated with silver rather than exhibiting any tendency to retain a fine neurofibrillary structure. (They are totally dissimilar to the metachromatic bodies in human and horses' brains described by Marion Smith [1949].)

Silver impregnation methods (Hortega's and Penfield's modifications) on frozen sections were used for demonstration of neuroglia (also the phosphotungstic acid method on paraffin sections). Some moderately good preparations were got after reimmersing the tissue blocks for some days in formalin-ammonium bromide solution. It was difficult to get any first-class astrocytic pictures after Cajal's gold chloride sublimate method—which was not surprising, considering the time the tissues had been in formalin. It was impossible to obtain any good silver preparations of oligodendroglia, and their rôle in the process was undetermined; in the more severely damaged areas they disappeared with other structures in the tissue dissolution. Microglial reaction was evident by the numbers of *gemästete Glia,* but rod forms were not particularly prominent. Augmentation of astrocytes in the impaired white matter was apparent in Nissl sections; this was associated with fibrillary proliferation forming meshworks of glial fibrils in zones where myelin had been lost, but in which vessels, ground substance and axis cylinders (some with swellings) still persisted (Figs. 14-16). There was often an isomorphic gliosis around clumps of phagocytic glia. Scattered here and there, not inside "tubes," but in relationship to glial fibrils, were large, irregularly-shaped protoplasmic masses with fragments of chromatin or even an indistinct nucleus at one side; there might have been some confusion with the swollen axis cylinders, but their position also helped to identify them. They were thought to be perhaps large pathological glia (Fig. 4) figured in some medical publications (see Wertham and Wertham [1934]), but they might be swollen axis cylinders of non-medulated fibres.

The vessels in the softened areas (after Wilder's method), when compared with unaltered vessels in the opposite columns of the cord, appeared more numerous with more prominent thickened walls, due to denser argyrophil reticulum.

There was little tendency for the process to affect grey matter of the brain stem except apparently by contiguous diffusion of the process. In the cord, in proximity to lesions in the white matter, there was shrinkage, chromatolysis (with the nucleus pushed to one side), or complete loss, of the large motor neurones in the ventral horns. In more damaged parts there were occasionally minute foci of malacia comparable to those seen in white matter. There was no neuronophagia, and the axones and dendrones in the grey matter of the cord showed no torpedo formation.

There were no pathological changes in the neurones of the motor area, of the red nucleus, of the cerebellar grey matter and nuclei, or, at least, no changes which could not be ascribed to poor fixation.

In all cases there was reaction seen in the spinal meninges; approximate and beyond the lesions in the white matter of the cord there were pial infiltrations

by mononuclears, lymphocytes and eosinophils; this occasionally extended into the nerve roots and ganglia, and sometimes was intense in the thickness of the dura mater.

Histological changes identical with those described in the spinal cord were found in the foci located in the mid-brain of one case and in the other in the cerebellar white matter and pons; they require no further elaborate comment. (It was noted that at the extreme margin of the acute lesions in the cord there might be only a few minute vessels with a minor degree of cuffing [Fig. 9].) In these two goats little cord was available, but a few such minute perivascular cuffs were found in sections (cord), hence a more complete observation might have revealed that the lesions had been more disseminated than they appeared.

Discussion

The number of cases limits some of the deductions to be made about the nature of this neurological disorder of goats. The uniformity of the type of lesion and the predilection for attack on the white matter of the brain stem in five goats (one 1951 case) are leads for work. More pathological study correlated with clinical observations might answer obvious questions which could easily be raised. This will provide better appreciation of the extent of variability of the kind and distribution of the lesions, and will establish whether there are chronic phases and whether there may be recovery. There is no proof now that either of these possibilities exist, or that the disease can be relapsing, and care will be required to substantiate clinical data on this aspect with meticulous pathological examinations. It was noted that in four of the reputed clinical cases of "lumbar paralysis" no nervous lesions were found to account for the symptoms. There are pointers that this "lumbar paralysis" might be easily confused clinically with maladies in which the animals might be unable to rise and walk, but which might not represent neuroparalysis. Such confusion need not be regarded as astonishing because of the difficulties attendant on neurological diagnosis of animal conditions. Even the meagre clinical data of these four goats (Cases 3, 5, 6 and 7) might indicate that three at least had suffered from another disease, perhaps parasitism; there was a history of "weakness" lasting months as opposed to the apparent subacute course of this disorder, regarded as a necrotising disseminated encephalomyelitis. There might be some danger of clinical perplexity, but there can be no pathological difficulties for there is no known nervous disease of goats with which it can be palpably mistaken. It would, however, be advantageous to possess more clinical information on the duration of the disease, on the condition of the tendon reflexes, and on examination of the cerebrospinal fluid.

It might be regarded as a patchy (fortuitous?), demyelinating disease, in the sense that there was an apparent proclivity for the process to destroy scattered areas of white matter in the brain stem. One might, however, draw attention again to the highly pertinent remarks of King and Meehan (1948) concerning the whole problem of demyelinating diseases and inflammation of the nervous system. According to them, there has been a leaning in medical

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neuropathological literature to entangle demyelination as a condition with it as a kind of disease, and a consequent tendency of including as a demyelinating disease, any disorder in which there has been destruction of white matter. There has also always been some bias to rather rigid views on what constitutes inflammation in the nervous system. It is unlikely that, in this goat condition, the primary effect is only on myelin; degeneration and loss is apparent, but this is not the sole prominent feature; the lesion is not highly selective demyelination in plaques, nor does it systematically affect tracts. This disease is more a form of patchy, necrotising encephalomyelitis (or malacia), in which myelin has undergone change and destruction in part with all other structures—ground substance, axis cylinders, and so on. In some parts, axis cylinders are more resistant to dissolution, and their reaction then produces unusual swellings, perhaps constituting a phase before final break-up. These axonal changes are seen in many unrelated neuropathological conditions of man, although axis cylinders have been a neglected item of observation in animal pathology; one might cite those described in human conditions such as disseminated sclerosis (Dawson, 1916), amaurotic familial idiocy (Globus, 1942), the Landry-Guillain-Barré syndrome (Haymaker and Kernohan, 1949), and they are a manifest feature in forms of cortical cerebellar atrophy (Norman, 1940) and also typical of secondary degenerations. (There are also magnificent descriptions of changes in axones and dendrones in the classical treatise of Cajal [1928]* on degeneration and regeneration of the nervous system, but we know little more about what they mean since his epoch-making discoveries.) Wertham and Wertham (1934) give figures of this same lesion, and state that the possible kinds of alterations in axis cylinders must be limited, and that no kind of described change is specific for any particular pathological condition. One might be tempted to regard the swellings as the inevitable response of the axis cylinder when the myelin sheath is destroyed and the fibres are left naked, but this cannot be truly so; they occur in axones and dendrones of so many different conditions, not all demyelinating or malacic, and are also seen in non-medullated fibres. They have been seen personally in other diseases in animals principally affecting white matter, but certainly not to the extent that they are prominent under low magnification with ordinary stains and confined to affected parts of white matter. (There seems to be no leaning towards belief by neuropathologists that a disease process can be initiated in axis cylinders, although Leiner (1922) mentioned that Wohlwill showed that in pernicious anæmia the axis cylinders might be primarily attacked.) There is no abundance of demonstrable lipoid from the breakdown of myelin in affected areas; this is not unique, for the same paucity is seen in some demyelinating diseases and might be related to tempo of a colliquative process. Comments on neuroglial response are reserved, yet there is evidence to indicate

* This is an authoritative book in which veterinary pathologists, beginning to take an interest in diseases of the nervous system, would find a wealth of valuable information. It was published in Spanish around 1914, but the work of the Spanish school did not become widely known for long after, and this English translation did not appear until 1928. It is a revelation to find that Cajal was using ammoniacal silver impregnations for axis cylinders in 1881—some 20 years before Bielschowsky made his classical contributions.

that both microglia and macroglia were involved in a way which is not peculiar.

The disease does not have any close parallel in human pathology; it is not a true transverse myelitis (in those goats in which only the cord was involved), nor is it a myelomalacia such as has been described consequent to vascular disorder of spinal vessels (see Russell Brain [1940], Davison and Keschner [1933], and Lichtenstein [1949]). Some remarks of Kinnier Wilson (1940) on subacute necrotising myelitis and Hassin and Bassoe (1922) on multiple degenerative softenings in man might suggest some points of similarity.

The paper by Emoto (1927) on the nervous disorder of goats (Japan) is unillustrated and the pathological descriptions are brief; even so, it might seem that the disease in Ceylon is similar, if not identical, in nature. Reasons were given why the claims of Emoto regarding a streptococcal origin might not be considered seriously; the lesions are unlike those which would be expected in streptococcal infections, and the only other paper on this caprine disease in Japan by Hashizume (1940) contradicts the views of Emoto. No data were obtained by Hashizume to indicate that the paralysis was infectious, or contagious, in origin. Some statements in the Muktesar reports imply the possibility that the same disease of goats and cattle exists in different parts of India. From the facts given by Delpy and Kaweh (1937), an enzootic neuroparalysis of goats has also apparently occurred in Iran, but the clinical and pathological data are too inadequate for any comparison to be made.

Crawford (1939 onwards) thought there might be some apparent resemblance to swayback in lambs (see Innes, 1950). There was no suspicion of cerebral demyelination in the goats studied personally, while the symmetrical secondary degeneration of the motor tracts in swayback is totally different from the restricted liquefactive necrotising lesions described. Some of the reports of Crawford also denoted that the disease occurred in adult animals, *never* in new-born, or young, kids, and that some affected pregnant goats gave birth to healthy progeny. These features all stand diametrically in contrast to swayback.

The initial experiences of Crawford eliminated the condition being due to an infective agent. If this disease in Ceylon is the same as observed in Japan by Emoto, then the later study of Hashizume (1940) harmonises with Crawford's negative findings. Nevertheless, the lesions are not completely dissimilar to those which might be expected in some form of specific viral encephalitis. They are distinct from the purulent form of acute meningo-encephalitis of sheep and goats caused by Listerella (see Gill [1931] and Graham, *et al.* [1943]). One would also require to draw the long bow in implying that there are analogous features with the lesions of the subacute Listerellosis of goats described by King (1940), which is the only detailed neuropathological account. In King's cases the granulomatous lesions were restricted to the brain stem, especially the spinal cord; there was myelin loss and mononuclear reaction, but the foci were related to vessels, and there was not the marked asymmetry of lesions as seen in the goats from Ceylon. *Listerella monocytogenes* was recovered from King's cases and were easily demonstrated by Gram's method in sections. On the face of it, therefore, it seems improbable that the Ceylon disease is a form of Listerellosis,

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yet there have been difficulties in isolation of this organism from animal conditions, and the complexity of producing disease of the nervous system experimentally by *Listerella monocyotogenes* is well known (as happened in King's cases). It is unlikely that parasites such as toxoplasma or encephalitozoon are connected causally, as far as their demonstration in sections are concerned, and it seems that they would have been discovered in the transmission experiments by Crawford. The lesions do not resemble those described for toxoplasmosis of the nervous system in man (see Paige, Cowen and Wolf [1942], and Frenkel [1949] for reference to the human disease, and Olafson and Monlux [1942] for reference to animal toxoplasmosis); they are unlike cases of toxoplasma encephalitis seen personally in dogs. There is thus some hesitancy in suggesting that further bacteriological and transmission study might still be profitable. In this event it would be essential to be certain that the donor inoculum of nervous tissue for transmission actually contained lesions, for the random selection of portions of brain and cord might result in negative results; this must be patent from the restriction in location and magnitude of the nervous lesions. Further, in experimental work of this kind it would be wise to confine initial projects to the natural hosts (goats), because we have many diseases in animals which are species specific.

Crawford considered that this paralysis might be compared in its symptoms, and in what he thought was cord degeneration (demonstrated by the Marchi technique), with conditions produced by experimental dietary deficiencies. There is now an immense literature on the effect of nutritional deficiencies on the nervous system (see Spillane [1947] and Follis [1948]), and only a few original references need be cited here. Mellanby (1931) described cord degeneration in vitamin A deficiency in experimental dogs (see also Mitchell [1941] on comparable work on pigs). Wintrobe, *et al.* (1936 and 1940) observed degeneration in the posterior columns and dorsal root ganglia and peripheral nerves of pigs which developed ataxia on a diet deficient in one, or more, of the components of the vitamin B complex. Ellis and Madsen (1941) produced locomotor incoordination with cord degeneration in pigs by dietary deficiency, but the exact responsible factor was not identified. Ataxia and motor weakness linked with cord lesions has not been indisputably established to occur naturally in domestic animals and proved to be due to any specific dietary factors (see Seifried [1940] and Innes [1950a]; there is no parallel in animals with subacute combined degeneration in man. The paper of Eveleth and Biester (1937) is of importance, for they could not incriminate vitamin A or the B complex in experimental pigs, or vitamins A, B complex, D or E in experimental dogs; consequently they suggested that there might be no correlation between the clinical paralytic signs and the cord degeneration, for the latter might be found without ataxic symptoms. There is thus only dubious evidence to associate any nutritional factor with this caprine nervous disease.

Reference was made to the negative results which attended the work done with poisonous plants by Crawford in Ceylon, and how he thought there might be some connection with lathyrism as described in text-books. A number of original articles on this "neurological" disorder were consulted, and disclosed that

there are no concrete data available which would enable any good comparison to be made, for the one essential aspect—knowledge of pathology—is entirely and strangely missing. This refers to the human disease and to the experimental projects which have been undertaken from time to time, while the pathological descriptions of lathyrism in domestic animals comes into the same bleak category. Consequently, lathyrism may still have to be considered as a possible cause of this caprine nervous condition. For reference to medical literature, see McCombie Young (1929), Filiminoff (1926), Manson Bahr (1940), Kinnier Wilson (1940), Rogers and Megaw (1944), and Shourie (1945); for pharmacological and experimental work, see Stockman (1929 and 1934); for reference to lathyrism in domestic animals, see Stott (1930), Petri (1930), Hutyra, *et al.* (1946), and Lander (1950).

Summary

1. A clinical neurological disorder of goats—lumbar paralysis—has existed in Ceylon for years. From the descriptions by Crawford, Mahamooth, Malkani, and Pillai, the onset may be acute or insidious, and a characteristic sign seems to be posterior ataxia and motor weakness progressing to complete paralysis.

2. A comparable disease may have occurred in Japan (Emoto and Hashizume), and perhaps also India (Muktesar reports), and Iran (Delpy and Kaweh), but more detailed pathological studies are required to establish this suggestion.

3. The nature of the lesion is that of acute inflammation with myelin loss, a perivascular and focal cellular reaction of lymphocytes, mononuclears, eosinophils and microglial and astrocytic response; in the more severely affected zones there is *status spongiosus*; persisting axis cylinders show irregularly globose, or elongated swellings, the significance of which is unknown; inclusion bodies are not present. The lesions are restricted in local magnitude and affect preponderantly the white matter of the brain stem. The process is regarded as a patchy, liquefactive necrotising encephalomyelitis and not as a demyelinating disease *per se*, and would account for neuroparalysis.

4. There is no evidence to incriminate, or eliminate, any particular ætiological agent, and further investigations are required with reference to the possibility that a neurotropic virus, bacteria, plant poisoning, lathyrism or nutritional deficiency might be implicated. There are emphatic differences between this nervous disease of goats in Ceylon and swayback of lambs in England or Australia, hence copper deficiency might thus be excluded.

Acknowledgments

Thanks are due to Mr. M. Crawford, M.R.C.V.S., Commonwealth Bureau of Animal Health, Weybridge, for much information on his own observations on the disease made when he was working in Ceylon. Without material from affected goats obtained through the courtesy of Dr. Mahamooth, Mr. P. G. Malkani, and Mr. C. Perumal Pillai, Peradeniya Laboratory, Ceylon, the study would not have been possible. The information provided by them is acknowledged in the text, and I am indeed grateful to all four workers for their generous help in all directions.

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Addendum (January, 1951)

Since the previous paper was written in the summer of 1950 attention was directed by correspondence to studies which were made, and published, in Japan during the war. These throw a different complexion on the whole problem. This note has been added so that there will be some immediate continuity between my own observations and with other facets of research which should be of importance to veterinarians, parasitologists, neuropathologists and workers in tropical medicine alike. The main purpose is thus to ring a bell on the possibilities of repercussions which arise from the Japanese work which cannot be ignored. Published data has been amplified in all directions by much correspondence and if the following facts had been known the original paper might have been written quite differently. In its original form it indicates the way of a pathologist studying an unknown problem of disease, but pieces of a jig-saw now begin to fit: suitable addition was made to the original title.

Yamagiwa, Shoho, Tanaka and others (1941-47) described a paralytic disease of sheep and horses in Japan (and Korea) in which there was a similar seasonal incidence to the disease of goats in Japan (and Ceylon). There was a comparable array of acute or subacute paralytic signs while the pathological lesions described, and illustrated, have all the hall-marks of an identical disease process to that seen in the Ceylonese goats. The salient features of the Japanese studies lie in the explanations of causation, for no similar ætiology has ever been established to cause nervous disease of man or animals. They contend that the microfilaria of *Setaria digitata* (natural host, cattle?) are transmitted by mosquitoes to sheep, horses and goats and finally invade the nervous system—the disease was thus named *Setariosis* (*Setariasis* might be better). Although the publication has been unobtainable so far, there is a Special Research Commission Report (Korea, 1939-1943) which apparently covers much work on the experimental side; perusal of this would be valuable. Correspondence has, however, filled in many gaps and if the pathological process is reconsidered in this fresh light of an helminthic cause then some obscure disappears. It would be important if confirmation of these results could be quickly obtained in regard to the cause of the caprine paralysis in Ceylon and it would be wise not to be influenced by ideas on what other filaria do in other tissues (e.g., in man or dogs). Examination of sections of the nervous lesions of Japanese cases has been made possible by the courtesy of the Japanese veterinarians. There is no doubt whatsoever in the author's mind that the same type of lesion is present to that recognised in the Ceylonese goats. This is, in effect, a focal colliquative necrosis complicated by a scattered secondary degeneration of nerve fibres with the bizarre swellings of axis cylinders, in, above and below the level of the lesion in the nervous substance. No doubt the filaria (undifferentiated larvæ or immature adults?) might cause some toxic irritation besides purely traumatic damage and account for the inflammation and meningeal infiltrations which contain many eosinophils. Helminthic parasites were not found in re-examination of the goat material from Ceylon but this might not be surprising for many reasons. The Japanese stressed that only one, or two, parasites

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might be present, they might wander away or die and disintegrate if the animal lived long enough. A search is apparently desirable in fresh acute cases and comments have already been made on the small magnitude of the lesion which might render it difficult to find in the first place. Random selection of nervous tissue might only reveal meningeal infiltration or nothing. In one series of Japanese cases in 176 sheep for example, parasites were found in 54 but a more meticulous examination of the nervous system raised this percentage. The crux of this problem might indeed lie in the necessity of examination of the *whole* nervous system and thus present a formidable task. In the end the fundamental proof of cause must lie in experimental production of the disease and this the Japanese claim to have performed. The blood of cattle containing microfilaria (*Setaria digitata*), as might be expected, was incapable of producing the disease by direct inoculation; however, microfilaria obtained after maturation in mosquitoes produced symptoms and lesions with parasites in the nervous system of sheep in about a month after inoculation into the skin. This is the same period which the Japanese claim elapsed after importation of sheep from Australia into Korea and until natural cases showed nervous symptoms. Some further clarification is required regarding the natural history of the development of the parasites.

The disease in Japan seems to be common enough as a seasonal enzootic in young horses, sheep and goats and it would appear to have been confused with equine encephalitis amongst other conditions—perhaps all grouped under the clinical medley of "lumbar paralysis." More meticulous examinations of the nervous system are required in these other vaguely described paralytic entities of animals in the Far East. Crawford's ideas on the similarity to the paralysis of "Kumri" in horses (India) might be particularly borne in mind for this sometimes has been associated with Setaria in the eye and paraplegia. Most text books on parasitology admit of great ignorance concerning the life cycle of *Setaria spp.* and what they do in natural, or unnatural, hosts and the latter might be the most important from the pathological viewpoint.

From what has been discussed in the main paper and this note, it is patent that serious attention should be given to the Japanese claims. In the case of the Ceylonese disease in goats all other leads regarding causation failed to produce any result. It is clear that the same type of paralytic condition might have occurred in domesticated, and wild, animals in many other parts of the world and has simply not been identified for easily understood reasons. Some highly suggestive descriptions have been found in the literature which need not be dealt with here. As a pointer it might be mentioned that a counterpart of the lesion has been seen in sections of a peculiar hæmorrhagic encephalomalacia in cattle occurring in the U.S.A., investigated originally by Jensen and Bracken (1950). Nor must we omit reflection that the same disease may have occurred in man (one might consider strange cases of eosinophilic meningitis with malacic foci of unascertained cause—e.g., Sheldon (1950). There are thus many avenues of work which can be exploited in a very positive direction and great credit must be given to the Japanese workers for their original observations and experimental studies.

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STUDIES ON THE ABNORMAL SPERMATOZOA OF BULL SEMEN

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(A) Introduction

MANY workers have examined innumerable samples of semen, but no widespread classification of the types of abnormal spermatozoa or their varying incidence has been published. Work described herein was in the nature of a survey, undertaken in order to afford such additional information, and, by providing a more elaborate classification, to further improve diagnosis of infertility.

Spermatology originated with the discovery of living cells in human semen by Ham and Leeuwenhoek about the year 1677 (Weismann, 1941). From the time of Retzius (1842-1919), much has been published, ranging from the linkage of abnormal spermatozoa with infertility, by Williams and Savage (1925), to the relationship of these abnormals to changes in the testicles, by Lagerlof (1934), and thence to the era of biochemistry, and to the use of the electron microscope (Bretschneider, 1947-1949).

In the study of spermatozoal deviation from the normal, much contradictory evidence exists. Thus, Williams and Savage, Lagerlof, Anderson (1939), Hotchkiss (1945), Lane Roberts, *et al.* (1948), all regard abnormal sperm as of importance, while Laing (1945) could find no correlation of abnormals to infertility. However, it has become increasingly clear that no single test will give really accurate prediction of fertility, and the study of semen by as many tests as possible is recommended (Hotchkiss, 1945; Haq, 1949).

For the purposes of ease of description, the sperm has been divided into three main regions, and in the pages following this sub-division has been used.

Region I comprises the entire head of the sperm, and consists of the anterior

vesicle (*Galea capitis*), protoplasm, and nucleus. Region II extends from the position of the anterior centrosome to the posterior centrosome (Maximov and Bloom, 1945). This comprises the sperm neck and the middle-piece, or body. Region III consists of the remainder of the tail, and extends from the posterior centrosome to the end. Hereafter this classification is used throughout.

Likewise, bulls that have shown good or satisfactory fertility rates are termed *normal* bulls: and the bulls with suboptimal fertility rates are termed *abnormal.* Bulls showing fertility rates between 1 and 65 per cent are termed *infertile*: the term *sterile* being restricted to bulls which are unable to reproduce.

The normal samples of semen have been taken from the bulls on farms during the course of other examinations. They have not been selected as especially good of their type. Thus the comparison is different to that of Haq (1948), who examined only samples selected by reason of their especially good characters as suitable for artificial insemination.

Hereafter, the term "Normal," when applied to either semen or bulls, indicates a satisfactory level of fertility. The term "Abnormal" has been used to cover all types of infertility and sterility.

(B) Materials and Methods

I. Material

Cases in which bulls were suspected of being the main cause of unsatisfactory breeding histories were notified to the laboratory by the attending veterinary surgeon. In every case the bull was examined by the writer at the farm on one or more occasions, so that details were obtained of all factors associated with the infertility. Where treatment was recommended, every effort was made to attend. Owing to thoughtlessness on the part of some farmers who failed to give any notice, a number of post-mortem examinations could not, unfortunately, be carried out.

The methods followed in the examination of infertile bulls were those of Haq (1949). On arrival at the farm, a general discussion was held between farmer, cowman, veterinary surgeon and the writer, special attention being paid to details relating to the breeding, early life, feeding and management, as well as the breeding efficiency of the bull. Some further discussion is, however, desirable on the determination of breeding efficiency.

Wherever possible, written records of the bull's performance were requested and examined. The records could be described as good on 21 farms, and as poor on eight farms.

For bulls of three years and over (59 per cent), the records were examined for the previous 12 months wherever possible.

Assessment of Breeding Efficiency

Assessment of records in the field and accurate evaluation of the fertilising capacity of semen present great difficulty. Firstly, many different methods of record keeping are in use, and some farmers record every service given by each bull, while others record incompletely, or allow two services to each cow and only record the number of cows served. Secondly, much variation exists in the repro-

ductive capacity of the cows and heifers in various herds, so that it is only in very large herds where two or more bulls are kept that the poor function of one bull is easily noticed. Thirdly, little uniformity exists in evaluation of fertilising capacity; thus, Anderson (1945), in his discussion on the subject, quotes a number of workers who expressed their results in the number of services per conception. In the development of artificial insemination centres the results are assessed on a percentage basis by a presumption of conception (Edwards, 1949).

In the present study, herds have been assessed as a whole, and fertility rates calculated on a percentage basis. This enables the inclusion of interesting cases from herds having very poor records, since it is always possible to obtain the number of cows in calf and those not in calf. When records of services were also available these have been collected and noted. Thus the fertility of cases examined have been calibrated on a scale of 0 (sterile) to 100 per cent (theoretical), according to the number of cows in calf or presumed in calf, and hereafter this percentage will be referred to as the fertility rate. As a consequence of infertility, herds frequently find themselves suddenly in a position where each cow has had three or more consecutive services and the number of services is vastly greater than the number in calf. The percentage of fertility rate may be compared with work expressed in numbers of services per conception by dividing the percentage rating into 100; thus, 20 per cent fertility equals five services per conception. When, however, the number of services to each animal is greatly increased, this approximation no longer holds good.

When assessing records, every effort has been made to obtain a reasonable picture of the fertility of the bull during the period of typical disease. Occasionally it will be found that if a fixed period of time is analysed a misleading impression of the bull is given. Thus, in 13 months ABR 34 served 45 cows with 98 services and left 22 in calf (48.8 per cent); but during the first six months of the year he served 32 cows and 22 conceived (68.7 per cent), while in the second period of the year he gave 66 services with no result (0 per cent). Thus, in evaluation, the longest possible period and the greatest possible numbers have been used to assess the fertility rate. The period, however, varies between two months and 12 months.

Cases Examined and the Incidence of Disorders

Twenty-nine bulls showing varying degrees of fertility, or other disorders, have been examined. They have been sub-divided as follows:—

GROUP I.—*Impotentia coeundi*		
(a) Congenital	1 bull	(3.45 per cent)
(b) Acquired	2 bulls	(6.89 per cent)
(c) Paunchiness	1 bull	(3.45 per cent)
GROUP II.—*Impotentia generandi*		
(a) Hypoplasia	2 bulls	(6.89 per cent)
(b) Seminal vesiculitis	1 bull	(3.45 per cent)
(c) Degeneration of testicle and/or epididymal abnormality	22 bulls	(75.87 per cent)

Of the bulls in Group I, two proved fertile on recovery, and semen samples obtained from them have been used together with 29 ejaculates from 10 other bulls of good fertility for comparison.

The bulls in Group II (25) were examined on 44 occasions, and 91 ejaculates of semen were collected. The bulls in Group II section (c) can be further sub-divided according to the types of abnormal sperm, as follows:—

Guernsey sperm decapitation	2 bulls	(6.89 per cent)
Atrophy	2 bulls	(6.89 per cent)
Semen containing predominantly abnormality of the sperm head	10 bulls	(34.50 per cent)
Semen containing predominantly abnormality of the mid-piece	2 bulls	(6.89 per cent)
Semen containing mixed abnormal sperm types	6 bulls	(20.70 per cent)

Ages of Abnormal Bulls

The greater proportion of bulls examined were young, 37.93 per cent being under 2 years old, 55.17 per cent being under 3 years old, and 72.42 per cent under 4 years of age. Sixty-three per cent of the first group and 60 per cent of the second group were sterile.

Methods of Examination

In every case, the bulls were examined on the farm. After consulting the animal's records, and inspecting his condition and surroundings, the collection of material was commenced.

The sheath was first irrigated and massaged after the method of Pierce (1947) with 250 c.c. sterile normal saline containing 2 per cent glucose, the apparatus consisting of a hollow metal tube 30 cm. in length, connected by rubber tube and air valve to a 10-oz. medicine bottle. This method proved advantageous in the writer's hands in overcoming as far as possible the contamination inherent in other methods of collection. In the case of quiet or moderately restive bulls, 80 to 90 per cent of the fluid was quite easily regained. On rare occasions, however, as in the case of very nervous bulls, a return as low as 20 per cent was obtained. During the administration, two men were required to steady the bull, one holding the rope or pole and the other bending the tail over the animal's back. The operator uses one hand to hold the metal tube in the mouth of the sheath and the other to massage the fluid into the sheath, thus making necessary the assistance of an attendant to elevate and lower the reservoir of saline.

On return to the laboratory, the washing was treated by the method already described (Haq and Rollinson, 1948) for the preparation of smears, for cultural examination, and detection of trichomonads.

As a routine, two samples of semen were then collected, using a fresh, sterilised cone and tube for each collection. So far as possible consecutive ejaculates were collected and, after recording the volume, 1 ml. (in the case of small quantities, half the ejaculate) was carefully poured into sterile glass test-

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tubes, plugged, and stored in ice for bacteriological examination. Estimations of the *pH.* and motility were then immediately made, using the methods described by Haq (1949). The semen sample tubes, after being wrapped in cotton-wool and four thicknesses of greaseproof paper, were then stored in chipped ice in a 2-pint Thermos jar.

Records were made at the time of the bull's libido, erection, and thrust at ejaculation. The first was obtained according to the method of Haq (1950), erection according to the time interval, and the thrust according to the following scale of five units:—

(i) Vigorous jump—bull's hind feet leave the ground, cone and tube being filled immediately;

(ii) Definite jump at ejaculation, but less vigorous than (i);

(iii) Slight jump at ejaculation;

(iv) Slight lurch at ejaculation, but no perceptible thrust; and

(v) No thrust—impossible to tell whether ejaculation has occurred.

Examination of the material was commenced immediately on arrival at the laboratory, i.e., usually within one to three hours. The semen tubes in their wrappings were removed from the Thermos and were carefully inverted three times. Semen was then immediately withdrawn from each tube, being used in the following way:—

(i) Two thin smears were prepared from each ejaculate by the blood smear technique;

(ii) 0.1 ml. was measured into 8 c.c. normal saline for density estimation, three drops of 1 per cent methyl violet 6B being added (0.147 to 0.15 ml.), and the dilution made up to 10 ml.;

(iii) 0.2 ml. was diluted with 0.8 ml. normal saline and used for the preparation of two flood smears of each ejaculate; and

(iv) 0.5 ml. was mixed with an equal quantity of opal blue-eosin for supra-vital staining.

The tubes, still wrapped, containing the remaining semen were returned to the Thermos, pending further examination at frequent intervals for motility.

Preparation of smears.—Direct smears were prepared from undiluted semen after thorough mixing of the sample. It was contrived to commence the smears about 20 mm. from the end of the slide and to limit them to less than 38 mm. in length. Smears were allowed to dry in air.

As a routine, one smear and one flood smear of each ejaculate were stained with Mayer's modified hæmalum (Haq and Rollinson, 1948).

Supra-vital staining.—In cases where the return journey to the laboratory was short and the examination could be commenced within two hours of collection, semen was stained by the opal blue-eosin method of Emik and Sidwell (1947). The solution comprised 12.5 per cent opal blue-Breslau and 0.5 per cent eosin in phosphate buffer *pH.* 7.2. Semen and the staining mixture were mixed in equal quantities (usually 0.5 ml.) in clean, dry test-tubes and allowed to react for 10 minutes before smears were prepared.

According to Emik and Sidwell, dead sperm stain blue by this method.

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The writer therefore estimated the proportion of living and dead cells after counting 300 sperm in the case of each smear, the figures thus obtained being used for comparison with the estimated motility of the semen after collection.

Preparation of tissues for post-mortem examination.—After collection of the reproductive organs at the time of slaughter and following on cultural examinations, representative portions of the testicles, the head, body and tail of each epididymis, the seminal vesicles and ampullæ were removed by means of a surgical scalpel and fixed in aqueous Bouin's solution. These were then dehydrated, embedded in paraffin wax, and sectioned at 5 μ. At the time of removal of tissue for fixation, smears were prepared from the cut surface of the testicles and from the contents of the epididymis and the ampullæ of the *vasa defferentia.*

In the case of both sections and smears, duplicates were always prepared, one being stained by hæmalum and eosin and the other by Heidenhain's hæmatoxylin.

(C) Types of Abnormalities and Method of Classification

(I) Types of Abnormalities

In considering the prediction of fertilising capacity of semen, it has become accepted by many workers that the larger the number of tests performed, the more likely is accuracy to be achieved (Hotchkiss, 1945; Lane-Roberts, 1948; Haq, 1948). As many tests as possible were undertaken by the writer, with special reference, however, to cellular abnormalities, which can be considered in two main categories, viz., tissue cells other than spermatozoa, and the sperm themselves.

(a) *Tissue cells*

Several workers have noted the presence of variously described tissue cells in semen (Williams and Savage, 1925; Pollak and Joel, 1944), and the work in this field has been reviewed and clarified by Haq (1948). In the present investigation, the methods of the latter author have been followed and the cells scored according to his recommendations (see (b) below).

(b) *Spermatozoa*

Retzius (1890-1921) made an important contribution by his studies of sperm, and his drawings of the variations of human sperm are rivalled only by the work of Lagerlof in the bull. Williams and Savage (1920) pointed out that infertile bulls showed larger numbers of morphologically abnormal sperm in their semen than fertile bulls, and since that time studies have been made by many others (Moench, 1931, 1932 and 1933; Pollak and Joel, 1939; Herman and Swanson, 1941; Hotchkiss, 1945). From their work, two schools of thought have emerged: (i) the biometrichians, who examine the variation in head length (Moench, 1932), and (ii) those who classify abnormal sperm on a general basis, using the total obtained as an index of the quality of the semen.

Certainly most work on abnormal sperm has been performed on fixed stained cells, and the varying methods employed may have affected the results obtained (Mercier and Salisbury, 1947). More recent workers have examined

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sperm diluted in normal saline (Laing, 1945), or fresh, on agar slides. These workers, however, still submit the sperm to abnormal stresses during dilution, at the same time losing the advantage of differentiation by staining capacity. The writer believes that by estimating the abnormalities in the fresh state on the hæmocytometer (i.e., the approximate percentage and predominating type), then scoring on a carefully prepared smear and, if necessary, checking by examination of flood smears, an accurate estimate can be made.

Variations in spermatozoa may take many forms, and very little agreement about the relative incidence of types is apparent. Generally speaking, spermatozoa vary from the normal firstly in respect of their staining capacity, secondly in their size, and thirdly in shape. The staining abnormality varies with the stain employed, from merely being darker or lighter than normal (Anderson, 1945) to eosinophillic or deep basophillic (own observation). Size varies from minute pinheads to giant cells of normal appearance and staining capacity (Lagerlof, 1934). Within these groups, variations in shape occur either in roundness of the head, posterior narrowing of the head (Williams, 1937), or anterior narrowing (Herman and Swanson, 1941). More rare abnormalities of the head include vacuolation, swelling or crenation of the anterior vesicle, abnormality of the *acrosome* (Teunissen, 1946; Hancock, 1949), or spontaneous shedding of the *Galea capitis* (Blom, 1945).

Middle-piece abnormalities include enlarged (swollen), filiform, beaded, shrunken, bent, duplicated middle pieces or abaxial attachment to the head. Also, protoplasmic drops may be associated with the middle piece. Their presence in semen is taken to denote immaturity of the sperm (Lagerlof, 1934).

Abnormalities of the tail are fewer in number, consisting principally of bends and coils (Herman and Swanson, 1941).

(II) Scheme of Classification of Abnormalities

(a) *Tissue cells*

Following the technique of Haq (1948), cells were scored in both the fresh state in the hæmocytometer, and, when fixed and stained, in flood smears. Only the simplest classification was used, morphology being the principal basis of differentiation. In cases which showed an exceedingly numerous cellular abnormality, e.g., leucocytes—ABR 32, these were scored in a similar fashion to sperm in the hæmocytometer. The types of cells noted were as follows:—

(1) *Nucleated and anucleated cells from the superficial layer of the prepuce.*—Large octagonal cells taking light to medium stain, sometimes studded with bacteria, debris or dead sperm.

(2) *Boat-shaped spermatogenic cells.*—Elongated cells with deeply-stained granular cytoplasm and indistinct cell outline, and containing one or more deeply-staining nuclei.

(3) *Spermatids.*—Mononucleate, multinucleate and anucleate. Regularly-shaped round cells about 10 μ in diameter with a deeply-staining nucleus were classified as mononucleated spermatids. Similar cells in which no nucleus was visible were considered as anucleated spermatids. Slightly larger cells containing

two to five small, deeply-staining nuclei were considered as multinucleated spermatids.

(4) *Leucocytes.*—These were identified by their size, staining affinity and lobation of the nucleus.

(5) *Spermatocytes.*—Larger cells with round or oval pale staining nuclei were regarded as spermatocytes. Difficulty was experienced in sub-dividing these cells into primary and secondary stages.

(b) *Spermatozoa*

When such an enormous population of individual cells is considered, possibilities of deviation from the ideal are exceedingly numerous. Previous classifications of the types of spermatozoa encountered have been either of the simplest nature (Laing, 1945) or of a minimum number of sub-divisions (Herman and Swanson, 1941). Typical examples are:—

	Lagerlof (1934)	Williams 1937)	Herman and Swanson (1941)
1	Differing from normal in size	Microsperms	Tailless
2	Double head or double tail	Megalosperms	Coiled tails
3	Abnormal-shaped heads	Pyriform heads	Pyriform heads
4	Loose heads	Defective acrosome	Other head abnormalities
5	Abnormal middle piece	Miscellaneous (head, body or tail)	Body abnormalities
6	Disturbed staining capacity		

Many of the more recent workers have been unable to demonstrate any relationship between the fertility rate and the percentage of abnormal sperm (see Laing, 1945), but none have considered whether different types of abnormals do in fact affect fertility. Tentative figures of the permissible numbers of abnormalities of the head, middle piece and tail, respectively, have been advanced by Lagerlof, discussed by Blom (1945), and endorsed by Haq, but no record is available of sub-divisions within these broad groups. In order to see if varying types of abnormalities influenced the infertility rate in different degrees, an extreme sub-division was devised by the writer. The divisions decided upon were guided by the work of Lagerlof (1934) and Haq (1948) in the bull, and Pollak and Joel (1942) and Hotchkiss (1945) in the human.

In considering sperm which deviate from the normal in any respect, the cell was considered in three parts according to certain anatomical divisions, namely, head, neck, and middle piece and tail. The sub-divisions decided upon seemed suitable from the practical standpoint of ease of recording data, coupled with the advantage of being flexible enough to encompass as many varying types as possible. The abnormalities of the head were regarded as of primary importance, those of the neck and middle piece of secondary importance, and those of the tail as of least significance.

(i) *Abnormalities of the head*

This table consisted of 59 separate sub-divisions. The first main division was into tailless and tailed heads, these in turn being sub-divided into normal

and abnormal. By this means, tailless heads showing abnormal staining capacity or morphology could be sorted out, and at the same time the ratio of tailless normal heads was known.

In the classification of abnormal heads, three main groupings were used, depending on the staining capacity of the sperm head, viz., eosinophillic, basophillic (normal colour), or hyperbasophillic (deeper than normal colour). The general principles guiding the allocation of abnormal sperm within these groups are set out below:—

Eosinophillic sperm heads (Figs. 1 to 3).—Heads showing a distinct red coloration were included in this group. Variation in both size and shape was often marked and frequently the only criterion as to whether the cell could be classified as a sperm head was the presence of a middle piece and tail, however rudimentary.

Basophillic sperm heads (Fig. 4).—This section included heads showing an exactly normal coloration by the standard method of staining, but varying in size and form. During the preliminary counting of the first 50 ejaculates, it became clear that a section would be necessary to include those sperm showing normal stain colour, normal size, but slight variation in form. Accordingly, in the final sub-division a further section was inserted.

Hyperbasophillic sperm heads (Figs. 5 to 9).—Heads having a deeper than normal blue coloration in any part were included in this group. Thus, heads showing extreme tapering, in which the posterior part only is deeply stained, come into this category.

Within each of these three groups, sub-divisions according to size were prepared, so that in each series, cells could be either larger than normal or smaller than normal. In the basophillic group a third sub-division was found necessary to include cells of normal size, this proving unnecessary in the eosinophillic and hyperbasophillic groups, where the great proportion of cells show marked variation from the normal in size.

The above-mentioned sub-divisions were finally split into eight sections, according to the shape of the sperm head, namely: pear-shaped (P) (Fig. 2), round (R) (Fig. 7), elongate (E) (Fig. 4), irregular (I), other shapes not fitting into these groups (O), vacuolated heads (Vc), double heads (D) (Fig. 5), and abnormal *Galea capitis* (V) (Fig. 12). The value of the sub-divisions chosen will be evident in Section D (below), where the numbers of "other" (O) heads are given. It will be apparent, however, that even within these eight groups some variation occurs, e.g., the extreme pinhead and the hyperbasophillic micro round head (Fig. 13) have to be classified in the same group.

Enumeration of abnormal heads.—Each ejaculate was recorded separately, the abnormal tailless heads being included in the total of abnormal heads. Tailless heads of normal staining and morphology were recorded as a separate group.

(ii) *Abnormalities of the neck and middle piece*

Neck.—The sperm neck was sub-divided into three sections, viz., swollen, kinked, or naked. Swellings of the neck were not easy to distinguish from

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swellings of the anterior part of the middle piece, while naked necks proved difficult to differentiate from slight mechanical damage.

Middle piece.—Division in this group was primarily determined by the presence or absence of pale protoplasmic drops attached to the middle piece. Sub-divisions of these groups depended entirely upon the morphology of the middle piece, the sections comprising bent, swollen, and kinked types, being once more sub-divided according to the position of the abnormality, whether anterior, middle or posterior. In considering, therefore, the type of abnormality shown in Fig. 19, as a bend of the posterior third of the middle piece, the writer has disagreed with earlier workers (Williams, 1943; Blake, 1945) who consider the abnormality to be one of the tail.

The classification entitled "swollen middle piece" requires some elaboration. Swollen middle pieces, with attached protoplasmic drop, denotes a pale staining drop surrounding the middle piece in any position (Fig. 20); whereas "swollen middle piece without protoplasmic drop" denotes darkly-stained irregular swellings, in which no differentiation could be seen between the swelling and the central fibril (Fig. 16).

Sub-divisions were also provided for coiled, double, thickened, and abaxial middle pieces, as well as for those with broken sheaths. A uniform darkly-stained middle piece was classified in the "thickened" sub-division group. "Double middle piece" includes only those cells showing a single normal head (Fig. 17) but with either a bifid or a completely separate double middle piece. Thus, all double-headed middle piece sperm are excluded from this group, as also are sperm with abnormal single heads but a double middle piece. The term "broken sheath" was devised to include all those cells in which only a single, bare fibril remained in place of the middle piece (Fig. 14).

(iii) *Abnormalities of the sperm tail*

Sub-divisions employed in this case were simple and straightforward, the primary division being that of "headed" and "headless" tails. The latter divisions were sub-divided into "double," "coiled" (Fig. 23), "bent," "kinked," and "tufted" tails.

(iv) *Other constituents*

Protoplasmic drops,—These were scored, when counting sperm, according to their staining affinity, in two groups: those showing a very pale eosinophil stain, and those taking a deep red stain. Previous workers have regarded these drops as being all of one kind, their significance being doubtful, although Haq found them to increase in number with frequent service.

In the writer's experience, free protoplasmic drops can be divided into two types, according to their staining characteristics and morphology (Figs. 24 and 25). Type A are pale-staining round eosinophil structures of fairly uniform size, being frequently granular in appearance. Type B stain deeply with eosin, giving them a prominent dark-red aspect. Commonly they are small and compact, circular or oval, and mostly about 2 μ in diameter. Much larger forms are also seen, ranging up to 12 μ in diameter, these larger forms being

slightly granular. The average size encountered in one bull (ABR 15), whose semen contained large numbers of the drops, was 6 μ in diameter (Fig. 25). When stained by Heidenhain's iron hæmatoxylin, the Type A drops take on a pale-grey coloration, while the Type B drops stain a deep grey or black.

.Medusæ.—The medusa bodies of Blom (1945) were scored on the flood smears, when making examinations for cells. They were recorded separately (Fig. 27).

(III) Methods of Counting Abnormalities

Recommendations for the evaluation of stained smears vary from those of Lägerlof, who counted 500 sperm on each of two smears, to Mercier and Salisbury (1947), who showed statistically that an experienced worker can obtain reliable results when only 100 sperm are examined. It was decided to follow the recommendations of Weisman and of Haq to count 300 sperm on one slide, this method giving a lower limit of detection of abnormalities of 0.3 per cent. A more searching examination was not deemed necessary in view of the comparisons later to be made with field fertility rates.

Smears were examined after uniform treatment, stained according to the method of Haq and Rollinson (1948), and the staining timed by stop-watch.

Since Mercier and Salisbury (1947) have been able to show that the experience of the operator in the counting may affect the accuracy of the examination, the first 50 ejaculates examined (containing both normal and abnormal sperm) were re-counted during the course of the investigation, only the re-counts being considered in the final results.

All smears were labelled at that end of the slide at which the smear commenced, all being placed so that this extremity was at the left of the microscope stage. Preliminary examination of the smear was made under 16 mm. and 4 mm. objectives, using a 25 mm. eyepiece in order to ascertain representative parts of the smear where the density was low and where there was no mechanical damage. Smears were examined by daylight in all cases, though occasionally, for special purposes, 150 candle-power Pointolite was used.

Smears were counted, using a 2 mm. oil-immersion objective (numerical aperture 1.3), fields containing 50 sperm or less being preferred. The number of sperm in the field was counted.

The abnormalities were then classified. Double heads were counted as one sperm, as also were tailless heads. Where the number of headless tails exceeded the number of tailless heads, the former figure was accepted. In general, all abnormal heads were recorded, regardless of the condition of the middle pieces and tail. Next, abnormal middle pieces with normal heads were classified; and, finally, abnormal tails with normal heads and middle pieces were recorded. The number of free protoplasmic drops was recorded as either pale- or dark-staining. Further fields were then searched until a total of 300 sperm had been examined. In cases of doubt about the grouping of particular cells, use was made of a 10 mm. eyepiece, giving an approximate magnification of 2,200.

If mechanical damage was suspected as the cause of any abnormality, the

sperm was considered normal as, for example, when a head and tail had become separated, but were lying in a straight line, the sperm being otherwise normal, no record was made. Abnormal tailless heads were included in the total of abnormal heads. In cases of Guernsey sperm decapitation (ABR 7, ABR 38), the same principle was applied, and after separation of tailed sperm from tailless sperm, abnormals were scored and recorded in the tables.

(To be concluded)

THE ISOLATION OF A NEUROTROPIC VIRUS FROM A DOG SUFFERING FROM THE SO-CALLED NERVOUS DISTEMPER

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Introduction

THE experimental production of nervous distemper in the ferret or the dog had not been reported in any previous communication. Many theories, hypotheses and observations had been in favour of the possible existence of a neurotropic virus or viruses closely associated with nervous distemper.

This preliminary paper will introduce the "Glasgow S123" virus which has been isolated from a clinical case of the so-called "nervous distemper"; its behaviour in different laboratory animals and its relationship with the classic Laidlaw-Dunkin distemper virus are described.

Material and Methods

History of the Case.

A male spaniel puppy, four months old, was reported to have had many nervous "screaming" fits. When examined at the West of Scotland and Glasgow Dog and Cat Home the temperature was 103.6 degrees F. The animal was slightly emaciated and showed slight purulent eye discharge with serous nasal discharge. A few days before the sudden onset of the nervous syndromes there was marked inappetance. The foot-pads, lungs and mucous membranes were apparently normal. The approximate duration of symptoms of illness had been one week.

Experimental.

The puppy was destroyed at the owner's request. Blood and the spleen were collected aseptically. The blood was allowed to clot and the serum taken for complement-fixation tests. The spleen was kept frozen on solid CO_2 for 48 hours until I returned to Weybridge from Glasgow.

On arrival, the spleen was emulsified and inoculated into a set of laboratory animals; 2 ferrets, 5 guinea-pigs and 6 mice. This set of animals is adopted whenever a new sample of suspected virus is investigated. In this way, an attempt is made to determine the presence or absence of the virus of lymphocytic choriomeningitis, with or without other viruses of the distemper complex.

PLATE I

FIG. 1
Early stage of the disease, 16 to 25 days after inoculation. Restlessness is marked.

FIG. 2
Back becomes arched (note clear eyes)

FIG. 3
Arching of the back more marked.

(*Article by Mansi, page* 214)

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PLATE II

FIG. 4
Head thrown backwards; forelegs stretched forwards.
Screaming begins at this stage.

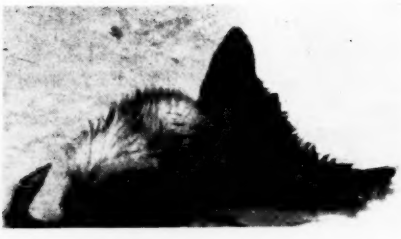
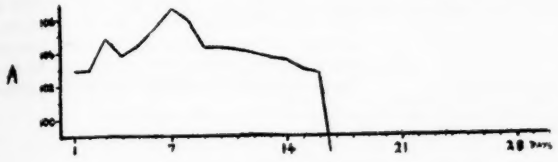


FIG. 5
Head further back and turning, legs now rigid.

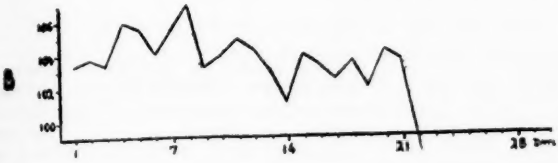
FIG. 6
Ferret now on left side with whole body rigid.
Death may occur at this stage.

(*Article by Mansi, page* 214)

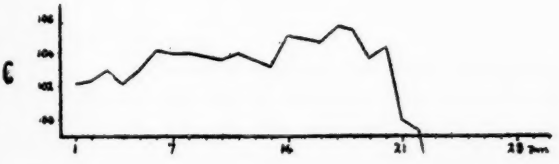
Text figures I of temperature charts of ferrets inoculated with different virus strains of the "distemper complex."



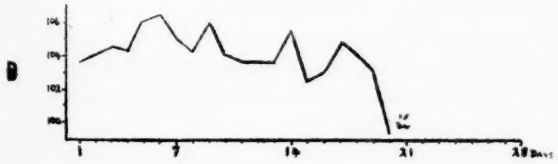
A.—Ferret No. 155, inoculated intracerebrally with "Glasgow S123," 5th ferret intracerebral passage. Definite thermal reaction developed on the 7th day. Animal remained in good condition until the 16th day, when sudden nervous screaming fits occurred, which caused the death of the animal in 2½ hours. No other lesions could be seen.



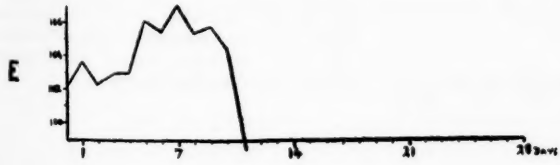
B.—Ferret No. 183, representing the 8th intracerebral passage, reacted thermally and developed typical screaming fits on the 22nd day after inoculation, and died in few hours.



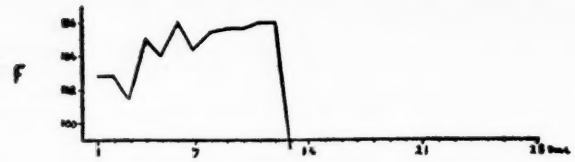
C.—Ferret No. 177, representing the result of intraperitoneal inoculation. This animal showed mouth lesions, purulent eye discharge, slight diarrhœa, and diminished appetite from the 14th day until the time of death on the 22nd day after inoculation.



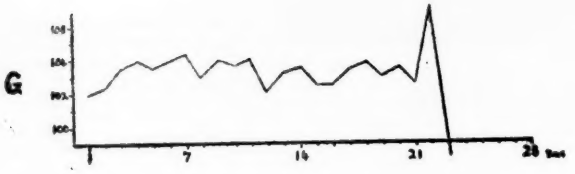
D.—Ferret No. 169, representing the result of inoculation of "Glasgow S123" virus, subcutaneously. Thermal reaction occurred on the 6th day. This ferret showed slight mouth lesions, purulent eye discharge and slight diarrhœa from the 14th day until it was killed on the 21st day after inoculation, when a nervous fit was seen.



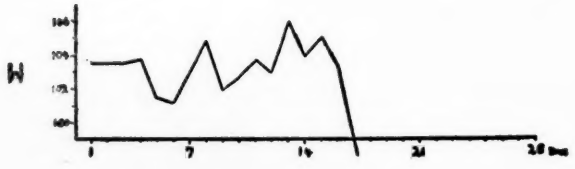
E.—Ferret No. 19/1949, representing the result of intracerebral inoculation of Laidlaw-Dunkin virus "W.D.V." A definite thermal reaction on the 5th day took place, followed by severe eye purulent discharge, mouth lesions and severe diarrhœa, which terminated by the death of the animal on the 11th day after inoculation.



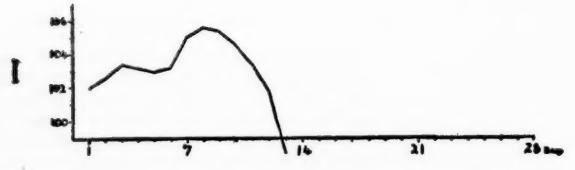
F.—Ferret No. 22/1949, representing the result of intramuscular inoculation of Laidlaw-Dunkin virus "W.D.V." A definite thermal reaction on the 6th day took place, followed by the classical symptoms of this infection in the ferret; purulent eye discharge, mouth lesions and severe diarrhœa. Death occurred on the 13th day after inoculation.



G.—Ferret No. 70, representing the result of intracerebral inoculation of Wellcome "hard pad" virus W.H.P., showed slight diarrhœa, no definite purulent eye discharge and absence of the mouth lesions, followed by death of the animal on the 23rd day after inoculation.



H.—Ferret No. 74, representing the result of intramuscular inoculation of Wellcome "hard pad" virus W.H.P. Symptoms similar to Ferret No. 70 occurred; death took place on the 17th day after inoculation.



I.—Ferret No. 140, representing the result of subcutaneous inoculation of Green distemperoid virus. Similar symptoms to Laidlaw-Dunkin infection in the ferret appeared. Death occurred on the 13th day after inoculation.

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The name "Glasgow" was given to this virus because of the fact that it was isolated in that city.

Microbiological examination technique excluded the possibility of bacterial infection.

Experimental Animals.

Ferret 104. This animal was inoculated intracerebrally with 0.3 c.c. and intraperitoneally with 5 c.c. of 1/20 emulsion of the puppy's spleen in 2 per cent normal serum saline (heated normal guinea pig serum). A thermal reaction developed on the 7th day after inoculation. The temperature dropped after two days and the animal remained in good condition until the 19th day after inoculation, when the temperature fell to under 97 degrees F. The animal was found dead on the morning of the 20th day. Apart from the temperature reaction, no other symptoms of illness had been noticed.

Ferret 105. Inoculation was carried out as in ferret 104. It developed a mild thermal reaction on the 7th day. Until it was killed, on the 13th day after inoculation, the animal maintained good health. The spleen and brain were collected on the 13th day in an attempt to recover the suspected virus. These organs were found to be bacteria-free.

No specific or diagnostic lesions were detectable on post-mortem examination of either of these ferrets.

Ferret Passage.

The brain and spleen collected from ferret 105 were emulsified separately and inoculated intracerebrally into two other ferrets. A thermal reaction took place on the 7th day after inoculation, the animals being apparently in good condition until the 20th day, when slight diarrhœa was noticed. On the 23rd day, the temperature dropped to subnormal and could not be recorded by the clinical thermometer. Suddenly, these ferrets showed a succession of nervous fits, which were all similar in character.

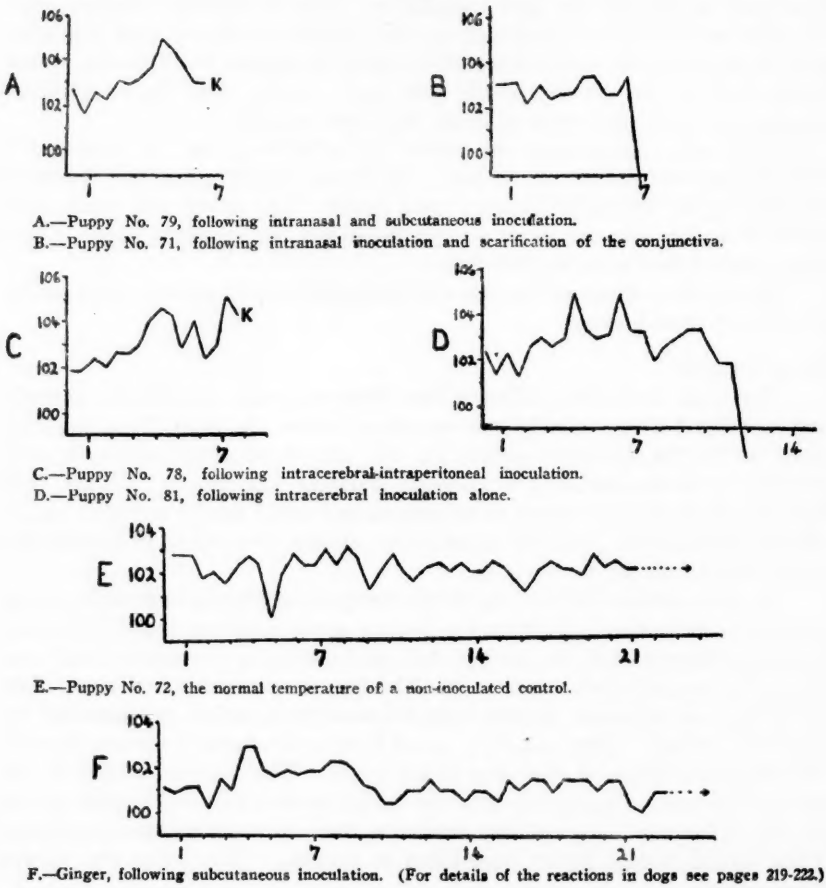
At the commencement of the fit the ferret showed restlessness with a jerky movement of the head. Respirations became accelerated and noisy. Then the head was thrown back, the back arched, and screaming commenced and continued for approximately 30 seconds. The legs were extended and rigid. The screaming and muscular rigidity were followed by a period characterised by muscular tremors. (Figs. 1, 2, 3, 4, 5 and 6 show the stages of nervous disorder following inoculation of this virus in the ferret.) The duration of the fit was about 2 minutes. During this time the animal pushed itself backwards against the side of the cage. A ferret may develop as many as 20 fits within 2 to 3 hours. These second-passage ferrets were killed *in extremis*. The brains and spleens were collected.

Post-mortem examination showed no specific or diagnostic lesions.

Later, in successive passages by the intracerebral-intraperitoneal inoculation technique, these fits have been reproduced in this typical manner for more than eight successive passages in the ferret. (The virus is still being passaged in this

laboratory.) Ferrets which have been left to die after the sudden appearance of these screaming fits have been found dead, after 2½ to 5 hours, in a peculiar position which suggested that death had resulted during one of these fits. In a few cases, presumably when the fits appeared during the very early hours of the morning, the animal was found after death with the back arched and the feet

Text figures II of temperature charts of dogs and ferrets inoculated with "Glasgow S123" virus (pooled brain emulsion; 2nd, 4th and 6th intracerebral-intraperitoneal ferret passages).

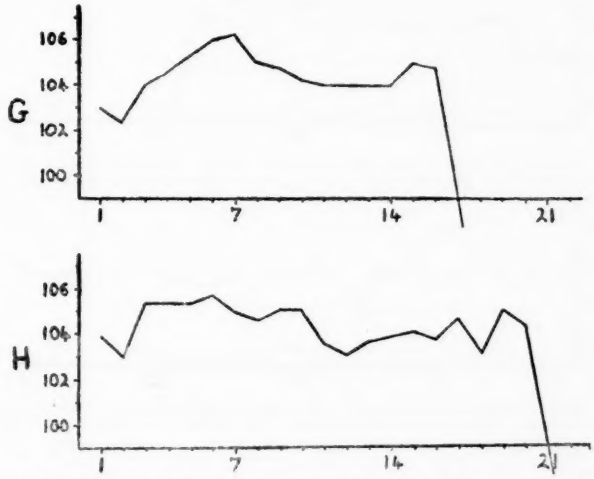


A.—Puppy No. 79, following intranasal and subcutaneous inoculation.
B.—Puppy No. 71, following intranasal inoculation and scarification of the conjunctiva.
C.—Puppy No. 78, following intracerebral-intraperitoneal inoculation.
D.—Puppy No. 81, following intracerebral inoculation alone.
E.—Puppy No. 72, the normal temperature of a non-inoculated control.
F.—Ginger, following subcutaneous inoculation. (For details of the reactions in dogs see pages 219-222.)

rigid. The teeth were firmly clenched, with lips retracted, the chin and part of the neck being moist, as if there had been profuse salivation. The sudden death of the animal occurring in the period ranging from the 16th to the 26th day after inoculation with this virus, together with the presence of the previously-described symptoms of fits, are considered characteristic of the infection.

After the fourth intracerebral passage in ferrets, brain or spleen emulsion from infected ferrets, inoculated by any route into another ferret, produced nervous fits. At this stage, however, other lesions, which were not observed or encountered in any ferret inoculated with the same material by intracerebral or intracerebral-intraperitoneal technique, appeared. Thus, slight mouth lesions, purulent eye discharge and slight diarrhœa, which resembled those encountered with the typical Laidlaw-Dunkin virus in the ferret, but in a milder form, were noticed.

It was noted that the route of inoculation was related to the resulting symptoms. The intracerebral-intraperitoneal inoculation technique gave almost



G & H.—Ferrets Nos. 180 and 181, representing the effect of the same inoculum in susceptible ferrets. Both developed typical screaming fits.

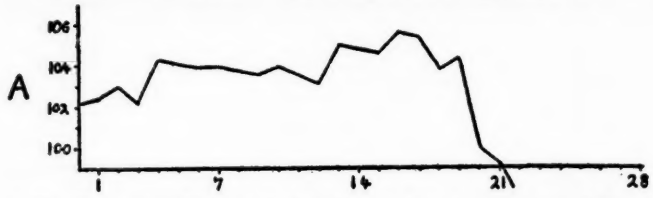
pure nervous syndromes, in particular the " screaming " fits, without any mouth lesions, purulent eye discharge or diarrhœa. On the other hand, continued subcutaneous inoculation resulted in the appearance of mouth lesions, purulent eye discharge and diarrhœa, with soiling of the perineal region, which were reproduced in successive passages in the ferret. Such visceral symptoms resemble, to a certain extent, those produced by the Laidlaw-Dunkin virus in the ferret, but differ in that the course is of longer duration, as none of the " Glasgow S123 " ferrets died before the 16th day after inoculation, while in Laidlaw-Dunkin infection death usually occurs at 11 to 13 days. In the subcutaneous passage of the " Glasgow S123 " virus, moreover, nervous fits are not uncommon. Mortality in ferrets after inoculation of the " Glasgow S123 " virus by any route in the ferret is 100 per cent. (Text figures I.)

In the Dog.

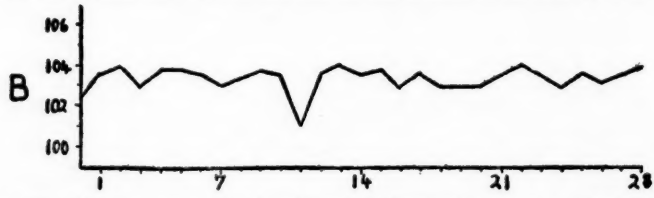
The inoculation of this virus into four susceptible puppies caused them to suffer from : fits, chorea, jerky movements of the head and neck, coma and death.

The first puppy, No. 79, a 19-week-old beagle, inoculated intranasally-subcutaneously with pooled 2nd, 4th and 6th intracerebral ferret passages, brain emulsion, developed a thermal reaction on the 4th day. In the evening of the 6th day after inoculation, fits began. During the period between fits the puppy walked in a circle, turning always towards the left. Periodically it fell over on its left side. Fits occurred whilst the dog was on its side and were characterised

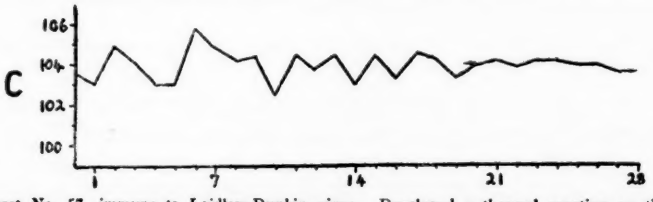
Text figures III show temperature reactions obtained when "Glasgow S123" virus was inoculated intraperitoneally into ferrets known to be immune to: Laidlaw-Dunkin virus, Wellcome "hard pad" virus, and Green distemperoid virus.



A.—Ferret No. 177, a susceptible control, showed mouth lesions, purulent eye discharge and slight diarrhœa, diminished appetite from the 14th day until the sudden death on the 22nd day after inoculation.



B.—Ferret No. 9/49, immune to "hard pad" and Laidlaw-Dunkin viruses. Developed neither a thermal nor clinical reaction. Resisted a further infection with another inoculum of the same virus.



C.—Ferret No. 57, immune to Laidlaw-Dunkin virus. Developed a thermal reaction on the 6th day for few hours, remained normal for at least 3 months. Resisted another infection with the same virus.

by opisthotonus, sometimes accompanied by extension and rigidity of the fore-legs. The hind legs were never seen extended. Rarely, screaming occurred during the period when the head was held back and forelegs extended. After the fit, tremor of the leg muscles was marked. This gradually wore off, and the dog would then get up and continue walking in a circle. The conjunctiva was injected. There were some spots under the chin, but these were not very definite. There were no catarrhal symptoms. The puppy was destroyed in the evening of that day.

The second puppy, No. 71, a 21-week-old beagle, inoculated intranasally and by scarification of the conjunctiva, with the same pool material, showed no

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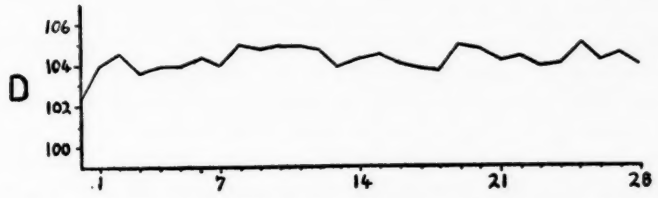
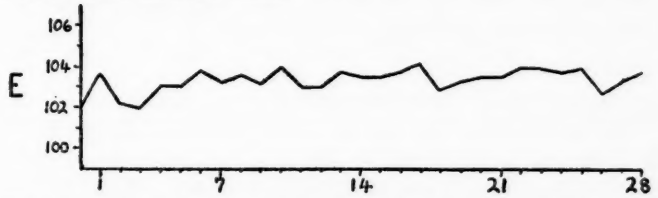
thermal reaction. It was bled for serum on the 7th day after inoculation. After being replaced in its quarters it showed one fit, which was followed by coma and death. The syndrome was probably precipitated by handling at the time of bleeding.

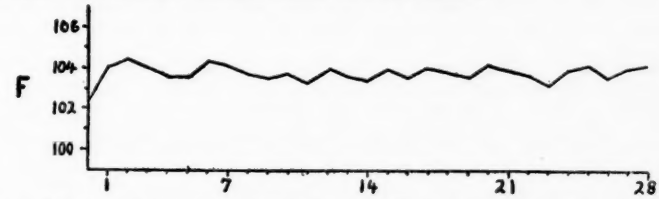
The third, No. 78, a 19-week-old beagle puppy from the same litter as No. 79, was inoculated intracerebrally and intraperitoneally. On the 8th day



D.—Ferret No. 159, immune to Green distemperoid virus, developed no definite thermal reaction, remained in good condition for over 3 months.



E.—Ferret No. 122, immune to Wellcome "hard pad." and to Laidlaw-Dunkin viruses. Neither thermal nor clinical reactions took place. Remained normal for over 3 months.



F.—Ferret No. 144, a ferret recovered from "hard pad" infection, found to be immune to Laidlaw-Dunkin virus. Developed no thermal reaction, and remained in good condition for over 3 months.

this dog was a typical clinical case of "chorea." Tremor of the leg muscles was very evident, and tremor of the jaw muscles caused the teeth to chatter. There was a little catarrh of the eyes and a suggestion of spots under the chin. A little foamy saliva was present around the mouth. The animal was destroyed in the last stage of the disease in the evening of that day. Having developed a thermal reaction on the 4th day reaching 104.6 degrees F., and another rise on the 7th day reaching 105.4 degrees F., the temperature dropped on the 8th day, when the nervous disorder took place.

The fourth puppy, from the same litter as Nos. 78 and 79, was inoculated intracerebrally only. It reacted thermally, the temperature being 105 degrees F. on the 4th day, dropping on the following day, and rising again on the 6th day, when serous eye discharge was observed. The animal was in fair condition

until the 11th day, when a peculiar nervous symptom developed. This was a sudden pulling of the head backwards and to the left. The jerk occurred every 5-10 seconds, and was followed by a slower movement of the head back to normal position. When food was offered, the puppy would put his nose down to the dish, but the frequently-repeated, involuntary withdrawal of the head made feeding impossible. In the late afternoon the right forelimb was exhibiting a jerky movement and the eye discharge became purulent. The animal was found dead on the following morning.

No hardening of the pads, diarrhœa or purulent nasal discharge was noticed.

Brain emulsion of puppies Nos. 78 and 79, when inoculated into two ferrets by the intracerebral-intraperitoneal technique, produced typical screaming fits, the ferrets dying on the 19th and 24th days, respectively.

When the ferret-passaged neurotropic virus was inoculated subcutaneously into two dogs, both over 2 years old and of unknown previous history, they developed a slight thermal reaction and slight diarrhœa from the 7th day to the 9th day, after which both dogs recovered. Both dogs were resistant to further infection with this virus. (Text figures II.)

In the Guinea-pig.

An emulsion prepared from the spleen of the original Spaniel puppy was inoculated into guinea-pigs Nos. 318 and 319 by the intramuscular and into guinea-pigs Nos. 320, 321 and 322 by the intracerebral routes. None of these guinea-pigs showed any thermal or clinical reaction. After 26 days their immunity was challenged with lymphocytic choriomeningitis virus (W.H.P.M. strain). They all reacted thermally and clinically, and were killed *in extremis* 7 days later. Fifteen guinea-pigs were inoculated by various routes with virus of different ferret passages on several occasions, producing neither a thermal nor a clinical reaction, and when challenged with lymphocytic choriomeningitis virus (Mc. or W.H.P.M. strain) all succumbed after showing the characteristic reactions and symptoms of L.C.M. infection in this species.

In the Mouse.

Mice Nos. 624, 625, 626, 627, 628 and 629 were inoculated intracerebrally (dose, 0.03 c.c. of 1/20) with the dog's spleen emulsion. They did not show any abnormality in condition over a period of 127 days. They were then challenged by intracerebral inoculation with lymphocytic choriomeningitis virus and they all succumbed on the 8th day after inoculation. Thirty-four mice were inoculated with this "Glasgow S123" virus from different ferret passages by the intracerebral route, on different occasions, without the production of disease. After several weeks all mice were inoculated intracerebrally with lymphocytic choriomeningitis virus, and all died.

In the Rabbit.

Rabbits Nos. 33 and 34 were inoculated intracerebrally with ferret 147 brain emulsion (fifth ferret intracerebral-intraperitoneal passage). Neither of the rabbits showed any thermal or clinical reaction during a period of 3 months' observation.

SUMMARY OF THE EFFECT OF "GLASGOW S123" VIRUS ON DIFFERENT LABORATORY ANIMALS

A. In Ferrets

No. of animals	*Route of infection*	*Reaction*		*Result*
		Thermal	*Clinical*	
6	I/C	4th to 8th day, usually 7th day after inoculation.	Sudden appearance of nervous screaming fits, 16 to 26 days after inoculation.	Death a few hours after the appearance of the fits.
13	I/C-I/P	Ditto.	Ditto.	Ditto.
8	I/P	Ditto.	Slight mouth lesions, purulent eye discharge, and slight diarrhœa, 10 days after inoculation. Nervous screaming fits usually occur 16 to 26 days after inoculation.	Ditto.
5	S/C	Ditto.	Ditto.	Ditto.
3	I/M	Ditto.	Ditto.	Ditto.

I/C = intracerebral. I/C-I/P = intracerebral and intraperitoneal. I/P = intraperitoneal.
S/C = subcutaneous. I/M = intramuscular.

B. In Dogs

No. of animals	*Route of infection*	*Reaction*		*Result*
		Thermal	*Clinical*	
1	I/C	4th day.	Jerky movements of the head and neck; purulent eye discharge appeared 11th day after inoculation.	Death on 12th day after inoculation.
1	I/C-I/P	4th day.	Chorea and fits on 8th day after inoculation.	Destroyed *in extremis* same day.
1	I/N-S/C	4th day.	Fits and chorea, 6th day.	Destroyed *in extremis* same day.
1	I/N Scarification of conjunctiva	No reaction.	Sudden fit, coma and death.	Death on 7th day after inoculation.
2	S/C	Slight.	Slight diarrhœa.	Recovered.

I/N = intranasal.

C. In Other Laboratory Animals

Animal species	*No. inoculated*	*Route of inoculation*	*Reaction*		*Result*
			Thermal	*Clinical*	
Rabbit	2	I/C	None.	None.	Survived.
Mouse	34	I/C		None.	Survived.
Guinea-pig	5	I/C	None.	None.	Survived.
Guinea-pig	2	I/P	None.	None.	Survived.
Guinea-pig	8	I/M	None.	None.	Survived.

DOSES

In ferrets, dogs and rabbits ... ... I/C = 0.3 c.c. of virus emulsion.
In guinea-pigs ... ... I/C = 0.1 c.c. of virus emulsion.
In mice ... ... I/C = 0.03 c.c. of virus emulsion.

Serological Investigation

(1) *Complement-fixation Tests.*

Serum collected from the original puppy S123 and from infected ferrets was tested against different antigens, including L.C.M. "Mc." (guinea-pig spleen antigen), Laidlaw-Dunkin distemper virus "W.D.V." (ferret spleen antigen) and Wellcome hard pad virus "W.H.P." (ferret spleen antigen). The spleens of ferrets, infected with these different viruses, were tested separately for the absence of L.C.M. virus by mouse inoculation test, and for specificity by ferret inoculations for the reproduction of the respective infection before being used as antigens.

The results of such tests proved the close antigenic relationship between the "Glasgow S123" virus and other members of the distemper complex (Laidlaw-Dunkin virus "W.D.V." and Wellcome hard pad virus "W.H.P."). No relationship between the "Glasgow S123" virus and lymphocytic choriomeningitis virus was observed. (See Tables I and II.)

(Details of the application of the complement-fixation tests to the study of the distemper complex and L.C.M. in dogs will appear in a separate paper.)

TABLE I

Some results of complement-fixation tests on the original puppy S123, serum and antigens prepared from infected ferrets with this virus and other known viruses of the distemper complex

Virus Strain ...	L.C.M. Mc				Distemper Cx. W.D.V.				Distemper Cx. W.H.P.				Saline (for anti-complementary effect)			
Animal Tissue ...	Guinea-pig Spleen				Ferret Spleen				Ferret Spleen							
Complement dilution 1/	50	70	90	110	50	70	90	110	50	70	90	110	50	70	90	110
Sera dilution 1/4																
Pup. S123	0	0	0	3	4	4	4	4	4	4	4	4	0	0	0	0
Pup. 19 norm. ...	0	0	0	tr.	0	0	0	tr.	0	0	0	0	0	0	0	0
Pup. 20 norm. ...	0	0	0	tr.	0	0	0	1	0	0	0	tr.	0	0	0	tr.
Rab. 14 L.C.M. ...	4	4	4	4	0	0	0	0	0	0	0	0	0	0	0	0
Rab. 10 W.D.V. ...	0	0	0	tr.	4	4	4	4	4	4	4	4	0	0	0	0
Rab. 11 W.H.P. ...	0	0	1	2	4	4	4	4	4	4	4	4	0	0	0	tr.
Rab. 33 Gl. S123 ...	0	0	0	0	4	4	4	4	4	4	4	4	0	0	0	0
Rab. 24 norm. ...	0	0	0	1	0	0	0	tr.	0	0	0	tr.	0	0	0	tr.
Saline	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Sera: All diluted to 1/4 and heated for 15 minutes at 65° C.
Antigens: All diluted to contain at least 3-4 antigenic doses.
Complement: Fresh or taken from the freezing box —20° C.
All test components, 0.25 c.c. amounts.
Pup. = puppy. Rab. = rabbit. 1/50 = complement dilution.
0 = complete hæmolysis. 4 = complete fixation. tr. = traces.

All sera of animals were collected from experimentally-infected animals with the exception of Puppy S123 (the origin of the "Glasgow S123").

TABLE II

Antigenic relationship of "Glasgow S123" and other viruses of the distemper complex group

Virus Strain ...	L.C.M. Mc				Distemper Cx. W.D.V.				Distemper Cx. "Glasgow S123"				Saline (for anti-complementary effect)			
Animal Tissue ...	Guinea-pig Spleen				Ferret Spleen				Ferret Spleen							
Complement dilution 1/	50	70	90	110	50	70	90	110	50	70	90	110	50	70	90	110
Sera dilution 1/4																
Pup. S123	0	0	0	3	4	4	4	4	4	4	4	4	0	0	0	0
Pup. norm. 19 ...	0	0	0	tr.	0	0	0	tr.	0	0	0	tr.	0	0	0	0
Pup. norm. 20 ...	0	0	0	tr.	0	0	0	1	0	0	0	tr.	0	0	0	tr.
Rab. L.C.M. 14 ...	4	4	4	4	0	0	0	0	0	0	0	0	0	0	0	0
Rab. L.C.M. 21 ...	3	4	4	4	0	0	0	0	0	0	0	0	0	0	0	0
Rab. L.C.M. 22 ...	3	4	4	4	0	0	0	0	0	0	0	0	0	0	0	0
Guinea-pig L.C.M.	4	4	4	4	0	0	0	0	0	0	0	0	0	0	0	0
G-p. Glasgow ...	0	0	0	0	2	3	4	4	2	3	4	4	0	0	0	0
G-p. W.D.V. ...	0	0	0	0	1	2	4	4	2	3	4	4	0	0	0	0
G-p. W.H.P. ...	0	0	2	4	4	4	4	4	4	4	4	4	0	0	0	4
Rab. W.D.V. 9 ...	0	0	0	3	4	4	4	4	4	4	4	4	0	0	0	2
Rab. W.D.V. 10 ...	0	0	0	tr.	4	4	4	4	4	4	4	4	0	0	0	0
Rab. W.D.V. 18 ...	0	0	0	0	3	4	4	4	2	4	4	4	0	0	0	0
Rab. W.H.P. 11 ...	0	0	1	2	4	4	4	4	4	4	4	4	0	0	0	tr.
Rab. W.H.P. 19 ...	0	0	0	3	4	4	4	4	4	4	4	4	0	0	0	tr.
Rab. Glasgow 33 ...	0	0	0	0	4	4	4	4	4	4	4	4	0	0	0	0
Rab. Glasgow 34 ...	0	0	0	0	2	3	4	4	1	3	4	4	0	0	0	0
Dog. L.C.M. 75 ...	4	4	4	4	0	0	0	2	0	0	0	0	0	0	0	0
Dog. L.C.M. 74 ...	4	4	4	4	0	0	0	0	0	0	0	0	0	0	0	0
Rab. norm. 24 ...	0	0	0	1	0	0	0	tr.	0	0	0	tr.	0	0	0	tr.
Rab. norm. pool ...	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

(2) *Cross Immunity Tests.*

(a) Between "Glasgow S123" and Laidlaw-Dunkin virus, Wellcome hard pad virus "W.H.P.," and Green distemperoid virus.

When the "Glasgow S123" virus was inoculated into ferrets immune to Laidlaw-Dunkin virus "W.D.V.", Wellcome hard pad virus "W.H.P." and Green distemperoid virus, no thermal or clinical reaction took place. Such animals continued in good health for over three months. Controls inoculated with the same test does reacted thermally and clinically, developing the characteristic nervous fits which were followed by death. (Text figures III.)

The result of these experiments indicated the close immunological relationship between some stock distemper viruses and the newly isolated virus, "Glasgow S123."

(b) Between "Glasgow S123" and lymphocytic choriomeningitis viruses. The animals chosen for these tests were mice and guinea-pigs.

When guinea-pigs and mice were inoculated with "Glasgow S123" virus, they showed no symptoms and remained in good condition until they were inoculated with lymphocytic choriomeningitis virus, when they all reacted both thermally and clinically and died. From the result of these tests, it appears that there is no immunological relationship between these two viruses.

Discussion

The condition "nervous distemper" has been recognised for a long time. This term is widely applied to various nervous syndromes which may take place during the course of field canine distemper infection.

Complications of the central nervous system, following distemper, are probably the most serious with which a clinician has to deal (Dalling, 1946).

Although the theory has been widely held that the nervous syndromes occur as a complication to distemper, practitioners have reported that it is not uncommon for brain symptoms to occur from the time of the primary temperature rise. It is, however, unusual in natural infection for a dog to start at this stage with one fit, and to proceed, in the course of the next few days, to have multiple fits and die (Bateman, 1946).

Epileptiform fits, convulsions, chorea and other nervous syndromes were observed in the Glasgow area (Weipers, 1948), in the Southport area (Hewetson, 1948), and in other parts of Great Britain. The symptoms described in such cases were experimentally reproduced in dogs and ferrets with "Glasgow S123" virus in this laboratory.

In Australia, a proportion of dogs suffering from systemic distemper develop symptoms referable to the central nervous system. In addition, such symptoms may develop in the absence of clinically-recognisable distemper (Steel and Whittem, 1950).

In France, in some animals, there were observed protracted febrile attacks which coincided with an excellent general state of the patient, but after the elapse of some weeks, nervous symptoms appeared. These forms were usually incurable (Gorét, 1950).

The possibility of the existence of a highly neurotropic virus, immunologically unrelated to the canine distemper virus, was reported by Cordy (1942).

These observations are valuable, and as a result of the isolation of the "Glasgow S123" virus, and its behaviour in the ferret and the dog, with its highly neurotropic affinity, some confusion as to the nature of "nervous distemper" has been cleared up.

On first isolation of this virus, temperature reactions were not as marked as those that occurred following a few passages in the ferret. In the dog, the temperature reaction, which lasts a few hours, may easily be missed. Because the inoculated animals remain in apparently good health, natural cases in dogs may not attract the attention of the owners. In peracute or acute cases in the dog the nervous symptoms appear suddenly, without the previous development of any other clinically perceptible symptoms, e.g., diarrhœa, purulent eye discharge, etc.

Dunkin and Laidlaw (1926) encountered nerve symptoms in experimentally inoculated dogs in about only 10 per cent of their cases. The symptoms that they observed were chorea, fits and epileptiform convulsions. Chronic nerve disabilities were never seen. In the ferret, on or about the 14th day following infection, or about the 4th day of the disease, nerve symptoms sometimes made their appearance. They were observed in about 7 per cent of cases, and

occurred only in those which presented mild general symptoms (in some cases nerve symptoms alone were noticed), or in animals which appeared to be improving.

At the present, the Laidlaw-Dunkin virus, by whatever route it is injected, produces distemper in the ferret with all its classical symptoms, but rarely or never with evidence of the involvement of nervous tissue (McIntyre, *et al.*, 1948).

From the previous results of Dunkin and Laidlaw and the results obtained by McIntyre, *et al.* (1948), on the same strain of distemper virus, one may conclude that the street distemper virus may possess a degree of neurotropism which may be decreased by subsequent parenteral passage in susceptible ferrets and dogs.

Other agents have been suggested as the cause of nervous syndromes (McIntyre, *et al.*, 1948). Among these is the so-called "hard pad" virus, which, when inoculated into ferrets, sets up a disease which bears some resemblance to distemper in this species. Despite the longer course of the disease, definite nervous symptoms have not been provoked in ferrets (McIntyre, *et al.* 1948). The so-called hard-pad virus, therefore, seems to be devoid of any necrotropic affinity, whereas from our experience with "Glasgow S123" virus we know that, with any route of infection with this latter virus, nervous fits in ferrets are almost constantly produced.

This newly-isolated virus has the ability to affect other tissues in the inoculated ferret. The virus-cell relationship can be altered and the neurotropic virus will produce visceral lesions if the virus is passaged a few times parenterally in the ferret.

Gorét (1950) suggests that adaptation of the canine distemper virus to the central nervous system can occur in the field, yielding a neurotropic virus which can only be isolated from the dog by intracerebral inoculation of the ferret.

The failure of the Laidlaw-Dunkin virus recently to provoke nervous symptoms may be explained on the basis that this virus has been passaged continuously in the ferret by routes other than the intracerebral. This has resulted in the development of a strain of high virulence for ferrets and producing entirely visceral lesions. The intracerebral-intraperitoneal technique of inoculation of street viruses would seem to be of great value in the isolation of strains, and prevents the loss of neurotropic affinity.

Serological methods were of value in confirming the biological tests used to elucidate the nature of the new virus. In the complement-fixation tests the serum of the original puppy, S123, fixed complement in the presence of antigens prepared from ferrets infected with Laidlaw-Dunkin virus and with Wellcome hard-pad virus "W.H.P." The close antigenic relationship of the last two viruses has been noticed for some time in our complement-fixation tests. Different spleens collected from ferrets experimentally infected with the "Glasgow S123" virus gave positive results with sera prepared from immune rabbits and ferrets, immunised with Laidlaw-Dunkin virus and with Wellcome hard-pad virus.

Cross-immunity tests gave parallel results. Ferrets immune to distemper

hard pad or Green distemperoid virus were immune to " Glasgow S123 " virus. Such immunity tests indicate the close immunological relationship, although in some biological tests, for example, after ferret inoculation, the course of the infections differ.

Rabbits, guinea-pigs and mice are not susceptible to infection with this virus. In this respect the virus resembles the classic Laidlaw-Dunkin virus.

(Details of immunity against the distemper complex, using various immunising agents, will be published in a separate paper.)

Summary

1. This paper records the isolation of a strain of distemper virus (" Glasgow S123 ") with very definite neurotropic properties.

2. Ferrets and dogs are susceptible to the infection, developing, after inoculation, thermal and clinical reactions. Nervous syndromes form the main characteristic of this virus.

3. Rabbits, guinea-pigs and mice are not susceptible to this virus by any route of infection.

4. In the ferret the thermal reaction takes place between the 4th and 8th day, usually on the 7th day after inoculation.

After intracerebral-intraperitoneal inoculation only nervous fits appear. These occur between the 16th and 26th days, and are followed by the death of the animal within $2\frac{1}{2}$ to 5 hours after the onset of the first fit.

After continuous subcutaneous or intraperitoneal passage in the ferret, mouth lesions, swollen eye-lids, with purulent eye discharge and diarrhœa appear from the 10th day, and the animal usually dies between the 17th and 26th day following the development of nervous fits.

5. In the dog, a thermal reaction usually commences between the 3rd and 6th day, and may last for one or two days, and sometimes only for a few hours. A diphasic thermal reaction is quite common. Mortality in susceptible puppies is high. However, two dogs over two years old and of unknown origin were found to be more resistant to the subcutaneous inoculation of virus. The duration of the disease is about 6 to 12 days. Nervous syndromes which were encountered among all fatal cases are described.

6. Serologically, the " Glasgow S123 " virus is immunologically identical with canine distemper virus " Laidlaw-Dunkin," Wellcome hard-pad virus, and Green distemperoid virus.

7. This virus is unrelated to lymphocytic choriomeningitis virus, as shown by animal inoculation tests, complement-fixation, and cross-immunity tests.

Acknowledgments

I would like to express my sincere thanks to Sir Thomas Dalling, who has made it possible for me to carry out this research programme. Also, I am indebted to Mr. J. R. Hudson for his advice and constant interest, to Mr. I. Lauder and his colleagues at the University of Glasgow Veterinary School for their help in sending materials; to Mr. A. B. McIntyre

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for the supply of different samples of Laidlaw-Dunkin virus and Wellcome hard-pad virus and sera. I wish to thank the outside staff of the laboratory for their good care in keeping and looking after the experimental animals in the various isolation places, and my assistants, Mr. M. Abbott and Mr. P. Tilbury, for their help, and, lastly, my appreciation to Mr. P. G. Millar for assistance in the preparation of this paper.

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RHEUMATISM OF THE SHOULDER: A STUDY OF A CASE IN A HORSE

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Introduction

SHOULDER lameness seems to be uncommon in horses. The term is widely applied to affections of the regions of shoulder and arm, as the clinical picture is nearly the same.

This type of lameness may be due to various causes, such as rheumatic affections of the shoulder muscles and arthritis of the shoulder joint. The last-mentioned cause (arthritis) is hardly thought of, and most cases are attributed to rheumatics or more superficial lesions.

Herewith a record of a case of arthritis of the shoulder joint.

History

A brown, country-bred cart horse, about 12 years old, was admitted to the hospital of the college, and showed lameness of the N.F. limb. The main clinical features shown were pain on manipulation of the joint, and lameness that increased with exercise and decreased with rest. Nothing abnormal was observed in the muscles or tendons of the region. The horse had been treated for "rheumatics," but did not respond.

As the horse was old and not in good condition, it was destroyed and taken to the dissecting room, where the affected shoulder joint was studied carefully, and the follow findings were observed:—

The capsular ligament was much thicker than normal, and on examining

the interior of the joint, two distinct ulcers were found in the texture of the articular fibrocartilage lining the glenoid cavity of the scapula (Fig. 1).

The capsular ligament of the shoulder joint of the other limb was also somewhat thicker than normal, while the articular surface of both glenoid cavity and head of humerus of the same joint showed signs of chronic inflammation.

Examination of the other joints showed no abnormalities.

Examination was made in the dissecting room on joints of other horses with no previous history of shoulder lameness, and no such lesions were found.

Anatomy and Physiology

The joint is of the enarthrodial variety and it possesses only the capsular ligament in the form of a double-mouthed sac attached to both rims of the glenoid cavity above and the head of the humerus below, and the sac supports by its inner surface the synovial membrane of the joint, and so the binding action of the capsular ligament is more or less loose. On account of the absence of firm ligamentous union, the capsular ligament, together with the muscle's tendons—which take the place of lateral ligaments—permit a much greater range of movement between the articular surfaces.

During movement of the joint there is a tendency to the formation of a gap between the two articular surfaces. The flattened epithelial cells furnish fine villous processes, known as synovial fringes, to increase the extent of the secreting surfaces and to spread the synovia. The synovia contains traces of fat.

The function of the articular cartilage, when sound, is to furnish a very smooth, glistening surface to facilitate the playing of the two opposed articular surfaces during the movement of the joint. The articular surfaces are highly lubricated by the synovia, which not only shares the function of the cartilages, but also reduces to the minimum the heat supposed to occur as the result of friction between the two articulating surfaces.

The secretion of synovia becomes increased as movement continues.

This natural function becomes impaired by inflammation of the synovial membrane. The flow of synovia becomes increased, being watery at first, then thicker in consistency and less in quantity. Lack of oiling increases friction of opposed surfaces. This is a probable explanation of the erosion of the articular surfaces of this joint.

Acknowledgment

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I am also indebted to Dr. Kamal Seoudi, Assistant Professor, Radiology Department, Veterinary College, Egypt, for his kind help in preparing the photograph.

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PLATE I

FIG. 1

(*Article by Mohamed Fouad Said, page 229*)

OBSERVATIONS ON THE MODE OF GROWTH OF THE HAIR OF THE DOG

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Introduction

THE literature pertaining to the mode of growth of the hair of the domestic animals is very scant, and that work which has been recorded may be summarised as follows: (1) The hair pattern of various species has been studied by Kidd (1903) and by Wood-Jones (1924); (2) the periodic nature of hair growth in the rat by Butcher (1934), by Haddow, *et al.* (1945), and by Durward and Rudall (1949); (3) seasonal variations in the growth of the hair of the horse by Hayes (1903), of the Arctic fox by Pocock (1912), and of the horse, cow and donkey by Duerdon and Whitnal (1930); (4) the effect of various physical conditions on the mode of growth of the hair of the guinea-pig by Strangeways (1933 a, b), of the Merino sheep by Bosman (1935), and of the human beard by Eaton and Eaton (1937) and Kayser (1938); (5) the mode of growth of the hair on skin grafts in the rat by Butcher (1936, 1937); (6) the effect on the hair growth of a bitch suffering from hyperplastic endometritis by de Vita (1939) and of dogs receiving massive doses of œstrogens by Gardner and de Vita (1940); and (7) the classification of coat types according to the texture of the hairs by Robb (1946) and Joshua (1946), and according to the colour of the hairs by da Fonseca and Cabral (1945).

It is thus apparent that no significant studies have as yet been reported on either the mode or the rate of growth of the hair of the dog. The need for such a study became apparent when, in 1945, the "Dyed Greyhound" case was reported by Kirk. At the trial of this case it was stated that the hair of the greyhound grows at the rate of approximately one-half of an inch per month; a study of the literature regarding other species, however, suggested that the matter might not be as simple as this.

Wilson (1949) has stated that more knowledge on this subject would materially assist in the enforcement of the Kennel Club regulations concerning the dyeing of dogs' coats at shows.

Material and Methods

Two series of measurements were consequently undertaken on an entire, dark brindle, adult male greyhound housed at the Royal Veterinary College. The series which is presented below ran from 26.9.49 to 28.11.49.

The hair was clipped and carefully wet-shaved over an area 12 inches long and 2 inches wide, the length of the area being parallel to, and about 2 inches below, the spine. The reason for the choice of this site will be explained later.

Each seventh day about one inch of this area was reshaved (dry) with a hollow-ground razor, the shavings were collected and washed in absolute alcohol,

and, after being well shaken, a random sample was taken and mounted under a cover slip on a large slide.

The method of measuring consisted of the direct measurement of 100 hairs from each sample, using a microscope with a 3-inch objective and a × 5 eyepiece carrying a 10 mm. × 100 scale, which had been previously calibrated against a millimetre scale placed on the stage.

Results

The growth rates observed in the two series of measurements were essentially similar, but the standard deviation was smaller in the second series. Since it is considered that this was due simply to a general improvement in technique, only this series is presented.

Means of 100 measurements of hair length at 7-day intervals. 2nd series.

Date.	*Mean of 100 measurements.* (mm.)
26. 9.49	—
3.10.49	0.327
10.10.49	0.603
17.10.49	0.882
24.10.49	1.035
31.10.49	2.85
7.11.49	3.18
14.11.49	4.84
21.11.49	5.92
28.11.49	8.03

The mean lengths are plotted against a time scale in the figure. It will be seen that two distinct rates of growth of hair are demonstrated, an initial slow growth of about 0.04 mm. per day and a second more rapid growth period of about 0.18 mm. per day.

Discussion

In 1934, Butcher reported the phenomenon of cyclical hair growth in the rat, having found that at any one time certain hair follicles in the skin are active and others are in a resting phase, and that a wave of growth passes slowly over the body. This has since been most beautifully confirmed by Haddow, *et al.* (1945), who noticed a marked symmetry of certain hair reactions in the rat and photographed the advance of this growth wave after absorption of an alloxazine dye by the active growing follicles following intraperitoneal injection. They found, broadly speaking, that this growth wave passed from the mid-ventral line upwards on either side of the body towards the mid-dorsal line. Thus areas of skin on a line parallel with the long axis of the body would be expected to be in the same phase of growth at any one time. It was in anticipation of an analogous situation in the animal under investigation that the area of skin previously described was chosen. Haddow, *et al.*, reported, furthermore,

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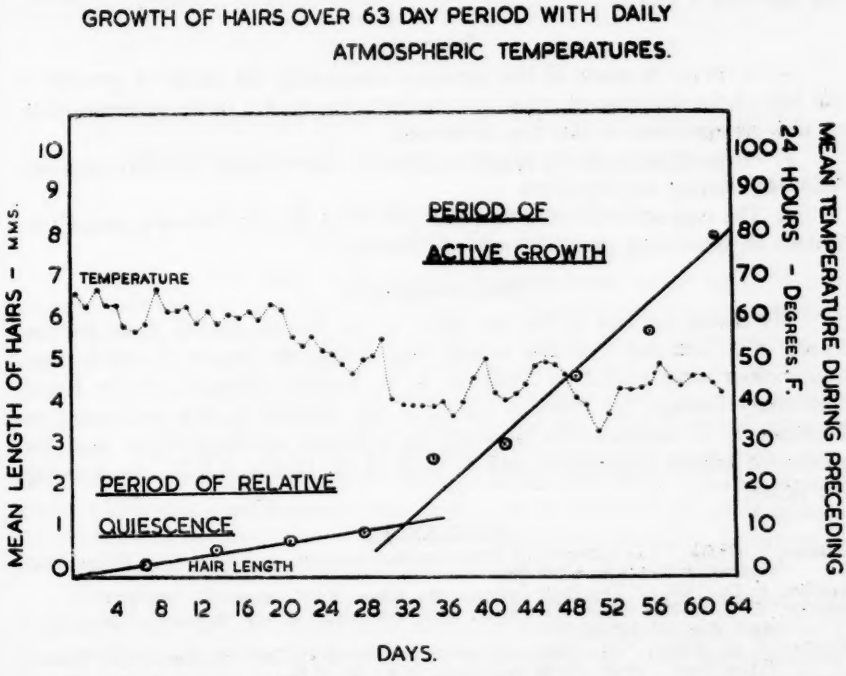
that these cycles of growth could be affected by factors such as nutrition, lactation, season and age.

Durward and Rudall, in a further study of factors which may affect the growth pattern (1949), state: "The mode of hair replacement varies in different animals and has a constancy in relation to species. Three different types of follicle activity determine the various patterns: (1) Continuously active, as in wool and human hair; (2) periods of activity and inactivity with neighbouring follicles in different phases; and (3) periodic activity with neighbouring follicles in the same phase."


GROWTH OF HAIRS OVER 63 DAY PERIOD WITH DAILY ATMOSPHERIC TEMPERATURES.
MEAN LENGTH OF HAIRS - MMS.
24 HOURS - DEGREES. F.
MEAN TEMPERATURE DURING PRECEDING
PERIOD OF ACTIVE GROWTH
TEMPERATURE
PERIOD OF RELATIVE QUIESCENCE
HAIR LENGTH
DAYS.

The results presented in the figure demonstrate two distinct rates of growth of hair in the dog, which we may reasonably assume to represent an initial phase of relative quiescence, followed by a phase of activity, of the hair follicles. It is impossible to be sure without further study whether the explanation for this is (1) that the period studied covered the end of a resting period and the beginning of an active period, or (2) that the growth picture recorded is the normal reaction to shaving. In view of the findings in other species, the latter seems unlikely. Also, in numerous preliminary trials on small areas ample evidence was obtained that shaving is followed by some degree of stimulation of hair growth and not inhibition. This is in confirmation of the findings of Seymour (1926) and Strangeways (1933), and in contra-distinction to the findings of Trotter (1928).

It would seem, therefore, that hair growth in the greyhound is probably in some manner a periodic and possibly a cyclical phenomenon, and suitable for inclusion in Group 3 of Durward and Rudall. Certainly the results obtained serve to stress the inadvisability of stating any dogmatic figure as representing the rate of growth of hair in this species with our present knowledge. We must first learn far more concerning the hair growth pattern, the factors which can influence this pattern, and the mechanism of moulting in the various breeds.

Such investigations are extremely tedious, however, and require the use of a large number of animals which may be observed continuously for a period of not less than a year.

Summary

1. A review is made of the literature concerning the mode of growth of the hair of the domestic animals, and the importance of a better understanding of this phenomenon in the dog is stressed.

2. Measurements of the length of hairs of a greyhound at 7-day intervals following shaving are described.

3. The suggestion is made that hair growth in the dog follows a periodicity pattern of alternating phases of rest and activity.

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THE CONTRIBUTION OF THE PHARMACEUTICAL INDUSTRY TO VETERINARY MEDICINE

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IT has been well said: "Wisdom is knowing what to do next; Skill is knowing how to do it, and Virtue is doing it." There may be many who possess all three of these desirable attributes in some measure, and if the quotation be used to illustrate three phases of the development of the modern veterinary product, it is not to suggest that those who play their part have either a monopoly of the one or a deficiency of the other two. Let us pay tribute to the wisdom of those men and women research workers whose labours enable us to know what to do next to solve the problems of our day; to the skill of the pharmacist, and others within this great industry, who know how to manufacture and distribute the products of research; and to the veterinary surgeon, who alone, by virtue of his training and experience, can give effect to the work of the others.

The record of service of the pharmaceutical houses to the profession is an honourable one and in a number of instances many generations of veterinary surgeons have been served with unbroken continuity by the same firm—Willows Francis was founded in 1751, Wyleys some time prior to 1757, and Hewlett's in 1832, to name but three.

The Traveller

Not all of us are old enough to remember, from our personal experience, the traveller who called on behalf of such firms in bygone days. This was before the word "representative" had been thought of, and, indeed, it would not have suited him at all, for he was an ambassador, no less, and his object was first to establish friendly relations with his client and then become the well-loved friend of the entire family. Business was not neglected, for his loyalty to his firm was implicit, but it took place at the proper time, which was the end of his visit. The contents of the shelves would be rapidly surveyed by his experienced eye

and the order made out for exact quantities to fill the containers to the top, a few suggestions would be made for additions to the order, which were usually agreed to, and he would make his farewells and be off. He was greatly respected and will be long remembered, not only for the service which he rendered, but also for his knowledge and integrity as a man of business. His modern counterpart, the veterinary representative, living in a less leisured time, has a different function to perform, but his knowledge of the preparations of his firm is large and the service which he can give the busy practising veterinary surgeon to-day is no less valuable.

With the development of pharmaceutical machinery to a high standard, many products, such as boluses, electuaries and drenches, formerly made up by the veterinary surgeon himself, were offered ready packed by the drug houses in a form which was both elegant and convenient. Drug lists grew larger as new preparations made their appearance for a host of different conditions in an increasing number of subjects, and competition between the houses stimulated research in order to make the best possible products and to evolve new ones.

Literature

There are now special departments in many of the larger firms where information is gathered together from the reports of research workers the world over, for it is necessary in commerce to keep abreast of what others are thinking and doing. Full use is made of such infomation in developing their products and in the preparation of descriptive literature, by which high-sounding name advertising matter is now called. Let it be said at once that the name is not undeserved, for much of it is well written, in addition to being informative, accurate and attractive. Often it embodies the results of many hundreds of experiments, carried out within the organisation of the firm issuing it, to determine such things as the toxicity, potency, the most suitable dilution for practical use, contraindications, and dosage. If the references to original work quoted in it are not sufficient, or if there is any point of doubt in the mind of the veterinary surgeon who wishes to use their product, it is part of the service given by pharmaceutical houses to give full answers to such enquiries.

It must be admitted that these services are not appreciated by all professional men, like the writer of a recent letter to a medical journal who refers to his amazement at " the ingenuity of the dispenser, the cunning of the manufacturer of fancy packings, and the erudition of those obscure pamphleteers who write the accompanying advertisements . . . the exotic names of those mentioned at the end . . ."

The preparation of literature is not usually undertaken until the results of extensive clinical trials have shown the advantages and limitations of the product and until the firm is fully satisfied that, from the original findings of the research workers, a formula has been developed which retains the active principles during a reasonable shelf life, and which is both effective and safe when administered in the doses recommended.

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Research

In the extensive sphere of interests covered by the larger pharmaceutical houses a comprehensive research programme has long been recognised to be of paramount importance, and very large sums are allocated annually for the provision and maintenance of the necessary laboratories and equipment. The industry is therefore able to attract a vast team of men and women scientists, many of them pre-eminent in their own field, and with their aid is able, not only to control and develop existing products, but also to engage in fundamental research. Great advances in medicine have resulted, particularly during the last fifty years, and veterinary medicine, being an essential part of the whole, has benefited in like degree.

When Ehrlich, in 1902, completed that remarkable series of chemical syntheses which resulted in Salvarsan, it was in the Bayer laboratories that the compound was first made, and from this beginning the science of chemotherapy has claimed the attention of research workers on an increasing scale and with far-reaching results. Other products introduced by the firm of Bayer include Novocain local anæsthetic, Evipan Sodium which was the first of the intravenous anæsthetics, Naganol for the treatment of trypanosomiasis, and Prontosil, the first of the sulphonamides.

In the laboratories of Parke, Davis & Co. half a century ago the pure crystalline hormone of the adrenal medulla, Adrenalin, was isolated and its chemical formula established. Later Pituitrin was introduced, and in 1927 the oxytocic and pressor principles of the posterior lobe of the pituitary were separated and standardised extracts prepared. Recently the Parke, Davis workers isolated Chloromycetin after some 2,000 moulds cultivated from 6,000 samples of soil, had been subjected to selective tests. This discovery was followed by the determination of its chemical structure and the firm was instrumental in achieving the synthesis of the antibiotic on a practical scale which has led to the manufacture of Chloromycetin by this method in substantial quantities.

The Wellcome Foundation is a unique organisation whose aim is the advancement of research in a very wide field, and many veterinary surgeons have met at conferences held at the research institution in Central London. In the laboratories at Beckenham, Dalling and Glenny added considerably to the knowledge of clostridial infections and were the first to produce, on a larger scale, products for the prevention of lamb dysentery. After Laidlaw and Dunkin in 1928 had evolved methods of protection against canine distemper, the task of adapting their experimental techniques to large-scale production was undertaken, and the very considerable technical problems overcome. The demand for biological products for protection against war-time diseases, both of humans and animals, rose to an unprecedented level and the size of the studs producing serum had to be increased threefold. In mastitis therapy the introduction of the single-dose collapsible tube for intramammary injections facilitated the administration of antibiotics and this type of container has been widely used. At the research station at Frant, in Sussex, there is an extensive

unit for the investigation of the diseases of farm animals, where field trials can be carried out under controlled conditions.

Outstanding contributions to veterinary science can be numbered among the achievements of the I.C.I. biological research laboratories. Foremost among these have been the discovery and development of Antrycide, a major advance in the treatment of prophylaxis of animal trypanosomiasis; Gammexane, and the more recent veterinary preparations of Lorexane, which occupy a pre-eminent position in the armoury of insecticidal weapons; and Sulphamezathine, the highly active, persistent and relatively non-toxic sulphonamide.

By investigating the therapeutic properties of a large number of sulphonamide derivatives, May and Baker workers were able in 1938 to evolve Sulphapyridine. This was at once recognised as a major chemotherapeutic advance and was followed soon afterwards by the production of Sulphathiazole in the same laboratories. An example of the company's interest in veterinary medicine is the acquisition of a farm at Ongar which was opened as a veterinary research station in 1945.

The production of penicillin on a commercial scale was a problem which arose during the last war and was solved by the co-operation of seven of the larger firms in the industry. In 1946 the Glaxo Laboratories were responsible for the introduction of the deep culture method of manufacturing penicillin, and are now operating a one-million pound plant for the manufacture of this and streptomycin. Aureomycin was discovered by the staff of Lederle Laboratories, a division of Cyanamid Products, Ltd., who have recently made arrangements for large-scale manufacture in this country.

Research by the Abbott Laboratories for a barbiturate which, from small dosage, would cause a suspension of cerebral activity and induce physiological sleep was rewarded in 1929 by the discovery of Nembutal. This is a product well known to veterinary surgeons and may be said to have revolutionised canine surgery. Evans Medical Supplies, Ltd., another old house, are well known to the veterinary surgeon for their biological products, etc.

Many other examples could be given of past achievements, as well as an impressive account of present endeavour, but in a brief survey significant omissions are almost inevitable; acknowledgment is made to the many who supplied much more information than it is possible to include. No attempt has been made at an historical record, or to assess the relative importance of one discovery over another, but rather to show some of the methods which have been adopted and the kind of resources which are made available by the pharmaceutical industry in the service of veterinary medicine.

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REVIEW

VETERINARY OBSTETRICS, by Professor Franz Benesch and Professor J. G. Wright. Pp. viii + 470, with 244 illustrations. London: Baillière, Tindall & Cox. Price 50s.

PROFESSOR WRIGHT says in his preface that when Professor Benesch's book was translated and published in this country in 1938 under the title "Veterinary Obstetrics" several useful criticisms were made by those who reviewed it. This is true and probably the most serious criticism that one could make of that book was that those who might use it as a guide would forget or never learn to use their hands, and would resort to embryotomy on almost all and every occasion.

Twelve years' experience has justified this criticism and in the case of our cows one is appalled by the number of occasions on which such serious surgical manœuvres as embryotomy have been resorted to when the intelligent use of the operator's hands, a couple of cords, a little patience and maybe a small pulley would have effected the relief of the mother and given hope of life to the fœtus. This is not a criticism of Professor Benesch's book *per se* for it was an excellent tome dealing with a specialised section of the obstetrical art, but rather that it stressed the grossly abnormal to such an extent that our young men were liable to forget that the great majority of difficult parturition cases could be resolved by much simpler methods.

Now in this book, whilst it includes Professor Benesch's original work, the subject is discussed on much broader lines and it can now rank as a more complete text-book on veterinary obstetrics.

It deals with the fœtus from its very conception until it is delivered into the world. In the first three chapters one is introduced to the œstrous cycle, pregnancy and its detection, and parturition—good sound material, for if one is to deal with the abnormal it is very necessary to possess a reasonable understanding of the normal. Following this maternal and fœtal dystocia and the causes thereof are discussed at length, and then a chapter is devoted to "The approach to an obstetrical case," probably the most important factor on any occasion when professional aid is required.

Chapter 11 is devoted to the use and value of epidural anæsthesia in the cow and mare, and chapter 12 deals with obstetrical equipment. As regards the latter it is pleasing to note that the instruments recommended are few and of simple design.

Chapters 15 to 21 deal in the time-honoured method with the delivery of the large fœtus and the correction of abnormal postures. These chapters are the best in the book and they are illustrated in such a manner that it is almost unnecessary to read the type.

In chapters 22, 23 and 24 embryotomy is discussed at length. These chapters give an excellent exposition of the uses of the chain or wire saw embryotome. Cæsarian section, which has now become almost a common-

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place operation in veterinary practice in the case of the cow and bitch, is discussed in great detail in chapters 28 to 29 and much valuable information is disclosed. The retention of fœtal membranes and the intra-uterine retention of the dead fœtus is discussed in considerable detail in chapters 30, 31 and 32.

Much information is given on ætiology and modern methods of treatment. We feel that these pages will be read with great interest by all veterinary surgeons, for the clearing of the post-parturient uterus has been the bugbear of practitioners for many generations. Any information which can help us to restore the uterus to a normal state and at the same time make the veterinary surgeon's job less distasteful is welcome.

Post-parturient hazards such as prolapse and perineal and vaginal injuries are described in chapters 33 to 34 and methods of surgical intervention are given in detail.

The final chapters are devoted to infertility and infections of the genital tract. Although emphasis is on the cow, the authors devote considerable space to the other domestic animals. Much of the information is the result of recent work. Many authorities are quoted and the authors have done a very good job in that they have made available to the ordinary man much knowledge which is not available to those who have not the time or facilities to keep up with the modern spate of publications.

Throughout the text of this book there are many excellent illustrations, the index is adequate, the format is good, and the authors and publishers deserve our thanks for they have provided, and within very reasonable compass, a text-book to which one may turn for information on almost any point which may arise in the theory and practice of veterinary obstetrics.

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